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Issue Date: 30 January 2003

Case No. 1999-LHC-1508
2000-LHC-1188

OWCP No. 5-105355
5-94575

In the Matter of

WILLIE M. RICHARDSON,
Claimant
v.

NEWPORT NEWS SHIPBUILDING AND DRY DOCK COMPANY,
Employer
and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party in Interest

Appearances:

Gary R. West, Esq., for Claimant
Robert E. Walsh, Esq., for Claimant
Hugh B. McCormick, III, Esq., for Claimant
Jonathan H. Walker, Esq., for Employer
Yusuf Mohamed, Esq., for Director, OWCP

Before:

RICHARD E. HUDDLESTON
Administrative Law Judge

DECISION AND ORDER

This proceeding involves two claims filed by Claimant, Willie M. Richardson, under the Longshore and Harbor Workers' Compensation Act, as amended, 33 U.S.C. § 948(a). (Hereinafter "the Act"). The relevant procedural history of these cases is as follows:

1. The Claimant filed a claim (OWCP No. 5-94575) with the Director, OWCP on February 16, 1995, alleging that he had contracted asbestos related lung disease

(hereinafter, "Asbestos claim") due to inhalation of airborne asbestos dust and fibers during his employment by Newport News Shipbuilding and Dry Dock Company. As a result, he claimed permanent disability, extent to be determined. No determination was made on this claim, which lay dormant at the office the District Director for several years until it was forwarded to this office for consolidation with a second claim. (See, file for Case No. 2000-LHC-1188).

2. The Claimant filed a second claim (OWCP No. 5-105355) with the Director, OWCP, on February 9, 1999, alleging that he had contracted severe chronic obstructive pulmonary disease (hereinafter, "COPD claim") as a result of "exposure to welding smoke, paint fumes-environment." He further indicated that it is not known whether his injury resulted in permanent disability. (ALJ 1)[1]

3. On March 18, 1999, the Claimant filed a form LS-18 requesting a formal hearing in his COPD claim on the issues of "Injury arising out of the course of employment" and "Temporary Total Disability." On April 12, 1999, the District Director forwarded the COPD claim for formal hearing. (ALJ 2)

4. On June 8, 1999, a notice of hearing was issued scheduling the COPD claim for formal hearing on September 21, 1999. (ALJ 3)

5. On August 23, 1999, Employer requested a continuance (COPD claim) or alternatively to have the opportunity to cross examine two physicians post hearing. (ALJ 4).

6. On September 8, 1999, the Claimant filed an additional Form LS-18 for his COPD claim, in which he indicated that the parties had recently received medical records indicating that he had reached maximum medical improvement in November 1998. Therefore, the Claimant added the issue of "Permanency." (ALJ 5).

7. On September 14, 1999, Employer filed a second request for continuance (COPD claim) or remand to permit development of evidence. Employer further requested permission to submit an application for § 8(f) relief, since permanency had now been raised. (ALJ 6). On September 17, 1999, the claimant responded by agreeing to a continuance, but objecting to remand, and citing § 702.321(b)(3).[2] (ALJ 7).

8. On September 21, 1999, an order was issued granting a continuance of the hearing

[1] The documents contained in the file for Case No. 1999-LHC-1508, are identified as ALJ exhibits, Claimant Exhibits in the COPD claim (CX), Employer Exhibits (EX) and Claimant Exhibits in the Asbestos claim as (CXA), Transcript of the July 18, 2000 hearing (Tr).

[2] A response was not filed by the Director to the motion. However, a review of the motion and response reveals that the Director was not served with a copy of either. At this point, no application for § 8(f) had been filed, and the Director had no reason to make an appearance in the case.

and rescheduling the case (COPD claim) for hearing on December 2, 1999. (ALJ 8).

9. Immediately prior to commencement of the hearing on December 2, 1999, the parties advised that a new issue had arisen regarding the application of § 33(g) of the Act to this case (COPD claim), although no LS-18 had yet been filed raising this issue. Further, the parties advised of the existence of the Asbestos claim pending dormant at the District Director's office (item 1 above, OWCP number 5-94575). (During an off record discussion, the parties agreed that a hearing should not be held at that time.)

10. On December 3, 1999 the Employer submitted a form LS-18 (COPD claim) raising the issues of application of § 33(g) and § 8(f) of the Act, and the nature and extent of disability, if any. (ALJ 9).

11. On December 7, 1999, an order was issued finding that the COPD claim and the Asbestos claim should be consolidated for a single hearing. Therefore, the December 2, 1999, hearing in this matter was continued, and the parties were ordered to submit pre-hearing statements which clearly and unambiguously identified all disputed issues. Further, the Director was ordered to forward the Asbestos claim (OWCP number 5-94575) for a consolidated formal hearing on both claims. (ALJ 10).

12. On December 14, 1999 (in response to the December 7, 1999 order) the Claimant filed an amended LS-18 (COPD claim), stating that "Claimant injured (sic) during the course of covered employment." Counsel stated the disputed issues were, "Permanent total disability benefits from 11/8/98 to the present and continuing, Medical treatment for Dr. Carlos Acosta and Dr. James Baker as well as any referrals made by the 2 physicians for the pulmonary problems, Medical causation."(ALJ 11).

13. On December 17, 1999, Counsel for the Director submitted a notice of appearance and a Form LS-18 (COPD claim) raising the issues of whether Employer is entitled to § 8(f) relief and whether the Director is entitled to the absolute defense of § 8(f)(3).(ALJ 12).

14. On December 27, 1999 an order issued (COPD claim) finding that the Claimant's statement that "Claimant injured (sic) during the course of covered employment" was just as ambiguous as the original LS-18, therefore did not comply with the December 7, 1999 order. Therefore, the Claimant was again ordered to identify the exact nature of the injury asserted.(ALJ 13).

15. On January 4, 2000, the Claimant filed a second amendment to his form LS-18 (COPD claim) and attached a copy of the original claim. The Claimant stated that "Claimant was diagnosed with pulmonary problems on 11/18/1998 due to his employment with Newport News Shipbuilding."(ALJ 14).

16. On January 6, 2000, an order issued (COPD claim), again finding that the most recent statement that "Claimant was diagnosed with pulmonary problems on 11/18/1998 due to his employment with Newport News Shipbuilding," was insufficient to clarify what injury was being claimed. Therefore the Claimant was ordered to state unequivocally whether he is claiming that his "severe chronic obstructive pulmonary disease" is due, in any part, to exposure to asbestos in his working "environment."(ALJ 15).

17. On January 17, 2000, Claimant responded (COPD claim) that he "is not claiming that asbestos exposure has played a role in his severe chronic obstructive pulmonary disease which has been diagnosed by Dr. Baker."(ALJ 16).

18. On February 14, 2000 the District Director forwarded a new form LS-18 (COPD claim) from the Employer dated February 2, 2000. At this time the Employer disputed the following issues: "Does § 33(g) bar the present claim when Claimant, without approval from the Employer, entered into a third party settlement for less than the claimed compensation in this case? Does § 8(f) relief apply based upon the Claimant's pre-existing asthmatic condition? Does the Claimant have an impairment due to 'severe COPD'."(ALJ 17).

19. On February 27, 2000, Counsel for Claimant advised that a settlement had been reached (COPD claim) and that an application for approval under § 8(i) would be submitted shortly.(ALJ 18).

20. On February 28, 2000, the Director forwarded the Claimant's Asbestos claim (OWCP No. 5-94575) (Item 1 above) and the Employer's form LS-18 disputing "Whether Claimant's attempt to withdraw claim for asbestosis is a proper purpose and in the interest of justice?"[3] (See, file for Case No. 2000-LHC-1188).

21. On March 19, 2000 Counsel for Claimant (Gregory Camden) advised that § 8(i) settlement (COPD claim) would not be presented and that the Claimant has requested that he be replaced by Attorney Robert Walsh.[4] (ALJ 19).

22. On March 30, 2000 a Notice of Hearing and Pre-Hearing Order was issued scheduling the two claims (COPD claim and Asbestos claim) for a consolidated hearing to occur on July 18, 2000. (ALJ 20).

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[3] Apparently Claimant's attorney in the Asbestos claim (Attorney Hugh McCormick, III) had filed a request to withdraw his Asbestos claim by letter dated January 5, 2000, addressed to the District Director. A copy had been faxed to this office by the Director on February 1, 2000. However, as that claim was not pending here, and the request was directed to the District Director, no action was taken by this office.

[4] Mr. Camden advised that the proposed settlement had been for $35,000, of which $5,000 would have been paid in attorney's fees. Further, he submitted a fee petition for $8001.50 plus expenses of $850.24.

23. On March 30, 2000, Counsel for the Director submitted his appearance in both claims (COPD claim and Asbestos claim) and a form LS-18, raising the issues of § 8(f) and § 8(f)(3). (ALJ 21).

24. On April 19, 2000, the Employer filed an unopposed motion for an enlargement of time to file a § 8(f) application (COPD claim). The motion was granted on April 20, 2000, extending the deadline to May 19, 2000. (ALJ 22).

25. On June 7, 2000, Counsel for the Director, by letter, advised that it was his understanding that no application for § 8(f) relief had been filed with respect to the Asbestos claim. Further he advised that it was his understanding that the Claimant was claiming compensation for temporary disability, and thus, § 8(f) was not implicated. (ALJ 23).

26. On June 21, 2000 Employer, by letter, advised Counsel for the Director that there is a diagnosis of permanent impairment and requested his position with respect thereto. (ALJ 24).

27. On July 18, 2000 a formal hearing was conducted on both claims. (See, Transcript). At the hearing, the Claimant was represented by Attorney Robert E. Walsh in his COPD claim and was represented by Attorney Hugh B. McCormick, III, in his Asbestos claim. Employer was represented by Attorney Jonathan H. Walker. Counsel for Director was not present.

28. At the formal hearing stipulations of fact were submitted and admitted into the record as Joint Exhibit 1 (Jx-1).[5] For his Asbestos claim, Claimant submitted three exhibits, CX 1A, 2A and 3A, which were admitted without objection. (Tr at 28.) For his COPD claim, Claimant submitted twelve exhibits, CX 1- CX 12, which were admitted without objection. (Tr at 29.) Employer submitted 31 exhibits, identified as EX 1 through EX 31, which were admitted. The only objection to the Employer's exhibits was to the second page of EX 6, which was admitted, but not for the purpose of establishing asbestosis from a medical standpoint. (Tr at 32, 34.) The record was held open 75 days for submission of briefs.

29. Upon commencement of the hearing, Mr. McCormick advised that he wanted to amend the claim for asbestos related disease. He stated that it was the Claimant's position that he was exposed to asbestos while working for the Employer from the "60's" to the "90's". As a result the Claimant contends that he has developed pleural plaques and pleural thickening, but that he does not have asbestosis. Thus, the Claimant amended his claim to seek only an award for medical monitoring under the Act. (Tr. 5-6).

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[5] The stipulations of fact were executed on behalf of the Claimant by his former Counsel, Mr. Camden. At the hearing, on the record, amendments to the stipulations were agreed to by Employer and Mr. Walsh with amendments (Tr. 21-26, JX 1). Mr. McCormick indicated that the stipulations were not relevant to the claim for medical monitoring.

30. On July 19, 2000, Attorney Gregory Camden filed a notice of lien (COPD claim) for attorney's fees and expenses. (ALJ 25).

31. On November 29, 2000, Counsel for Director filed a post hearing brief in opposition to § 8(f) relief (ALJ 26). The Employer filed its brief on November 29, 2000 (ALJ 27). Counsel for the Claimant (Mr. Walsh, COPD claim) filed his brief on November 30, 2000 (ALJ 28). On December 7, 2000, Counsel for the Claimant (Mr. McCormick, Asbestos claim) filed his brief (ALJ 29).[6]

32. During a telephone conference on March 12, 2001, the Claimant (COPD claim) and Employer were requested to provided additional argument regarding the application of § 33(g) to the COPD claim. Accordingly, on April 18, 2001, supplemental briefs were filed by Mr. Walsh (for Claimant) (ALJ 30) and Mr. Walker (for Employer) (ALJ 31).

33. Prior to issuance of a decision and order, on March 25, 2002, Counsel for Employer filed a motion to withdraw its stipulation made on the record (Tr. 55 and stipulation "11", *infra*) that asbestos exposure did not cause asbestosis and did not contribute to any lung impairment Claimant has. Additionally, Counsel advised that the parties jointly requested appointment of a settlement judge (by telephone) pursuant to 29 C.F.R. § 18.9. (ALJ 32).

34. On March 25, 2002, an order issued holding the case in abeyance pending settlement judge proceedings. (ALJ 33).

35. On May 9, 2002, the settlement judge issued a Notice of Conclusion of Settlement Judge Proceeding, advising the case had not been settled. (ALJ 34).

36. On June 28, 2002, an order issued lifting the stay of proceedings, and granting the parties 10 days to submit argument on the Employer's request to withdraw its stipulation that the Claimant has no impairment due to asbestos exposure. (ALJ 35).

37. On July 10, 2002, the Claimant filed an objection to Employer's motion, arguing that there would be incurable prejudice to the Claimant if the motion was granted. (ALJ 36).

38. On July 17, 2002, Employer filed a response arguing, *inter alia*, that the evidence submitted into the record indicated that the Claimant suffers from a mixed restrictive and obstructive lung defect, with no explanation other than asbestos exposure to explain the restrictive defect. Further, Employer argued that any prejudice to the Claimant could be cured by reopening the record to permit

[6] Three extensions of time had been granted for filing briefs, with the last deadline being November 29, 2000. However, no objections were filed to the timeliness of any brief.

additional evidence to address the issue. (ALJ 37).

39. On July 30, 2002, Claimant filed a response arguing that all of the information, medical reports and Court filings upon which the Employer relied in making its motion, were in Employer's possession and fully known at the time of the stipulation. (ALJ 38).

40. On August 22, 2002, an order was issued granting the Employer's motion to withdraw the stipulation; and the record was re-opened to permit the Claimant to submit any additional evidence he deemed necessary as a result, including (but not limited to) a new hearing, and/or taking Dr. Baker's supplemental deposition at the Employer's expense, if such was requested. The Claimant was directed to advise within 20 days how he wanted to proceed in the development and presentation of new evidence in response to the order. (ALJ 39).

41. On September 3 and 6, 2002, Counsel for Claimant advised that, pursuant to the August 22, 2002, order, Claimant would take the depositions of Dr. James Baker and Dr. Eric Freeman, within 30 days. (ALJ 40, 41).

42. On October 16, 2002, the Claimant submitted the deposition of Dr. James Baker, which is admitted as CX 13, and the deposition of Dr. Eric J. Freeman, which is admitted as CX 14.

43. Upon receipt of Claimant exhibits 13 and 14, the record in this matter is closed.

The findings and conclusions which follow are based on a complete review of the record in light of the argument of the parties, applicable statutory provisions, regulations, and pertinent precedent.

STIPULATIONS

At the hearing, Claimant and Employer submitted written stipulations of fact (JX 1 as amended at the hearing, see Tr. 21-26), which are accepted and adopted as findings of fact, with the exception of stipulation 11. The parties stipulated as follows:

1. That an employer/employee relationship existed at all relevant times;

2. That the parties are subject to the jurisdiction of the Longshore and Harbor Workers' Compensation Act;

3. That the claimant alleges pulmonary problems resulting from his employment with Newport News Shipbuilding and Dry Dock Company as diagnosed on November 18, 1998, by Dr. Acosta;

4. That a timely notice of injury was given by the employee to the employer;

5. That a timely claim for compensation was filed by the employee;

6. That the employer filed a timely First Report of Injury with the Department of Labor and a timely Notice of Controversion;

7. That the claimant's average weekly wage at the time of the injury was $820.20 resulting in a compensation rate of $546.81;

8. That the claimant has been paid no workmen's compensation disability benefits as a result of his injury, but he has received Sick and Accident Benefits in the amount of $5,200.00 for the period covering 11/17/98 through 5/17/99, and the Employer is entitled to a credit for these monies against compensation awarded for this period;

9. That the employer has not paid for medical services as required by 33 U.S.C. Section 907 (1998), however, medical treatment has been covered by the claimant's personal insurance with the [ ] having been made by the claimant [sic];

10. That the claimant has a wage earning capacity of $6.00 per hour for 40 hours per week from 5/18/99 and continuing.

11. During questioning of Dr. James Baker, at the hearing, the parties further stipulated that asbestos exposure did not cause asbestosis and did not contribute to any lung impairment he has. (Tr at 55.) However, by order issued on August 22, 2002, Employer was permitted to withdraw this stipulation. (ALJ 39).

ISSUES

The following issues are disputed by the parties:

1. Does § 33(g) of the Act apply to Claimant's claim for "medical monitoring" for asbestos related disease?

2. Does § 33(g) of the Act bar Claimant's claim for permanent total disability compensation due to occupational chronic obstructive pulmonary disease?

3. Is the Claimant entitled to an award for "medical monitoring" pursuant to 33 U.S.C. § 907(a) for asbestos related disease?

4. Is the Claimant entitled to temporary total disability and permanent partial disability compensation due to "non-asbestos related" occupational lung disease?

5. If the Claimant is entitled to permanent partial disability, is the Employer entitled to Special Fund relief under § 8(f) of the Act?

SUMMARY OF MEDICAL EVIDENCE

The following is a summary of the medical evidence admitted into the record:

A pulmonary function test performed at Sentara Hampton General Hospital on May 13, 1994, indicates a mild obstructive pulmonary impairment which was confirmed by increased RV. (CX 3-18). It also showed an additional mild restrictive pulmonary impairment. (CX 3-18). The person performing the exam stated that this opinion was subject to a physician's review. (*Id*.) *See also* (CX 3-19 thru 21)(EX 8).

Claimant's echocardiogram results were reported and read by Dr. M.D. Clark on June 1, 1994. Apparently unremarkable. (CX 3-17), (EX 14-13).

On a medical summary form of Employer it is noted that on June 24, 1994 Claimant was diagnosed with asbestos related lung disease. There is no signature. (EX 10-47).

In a letter to Dr. Acosta from Dr. Childs, dated June 24, 1994, he states that in his June 17, 1994 visit, Claimant was started on bronchodilators. Claimant stated, at first, that he did not notice much improvement in his shortness of breath and dyspnea, but on further questioning, Claimant did note some improvement. (CX 1-9). A lung examination revealed decreased breath sounds with no wheezes or rhonchi. *Id*. At that time, Dr. Childs' impressions were that Claimant had a severe obstructive lung disease with a mild restriction. *Id.* Claimant was told to follow up in one month. *Id*. *See also* (CX 3B-A), (EX 12-5).

In a letter to Dr. Acosta from Dr. Childs, dated June 26, 1994, Dr. Childs states that Claimant was seen initially on June 17, 1994 after six months of complaining of shortness of breath associated with increased dyspnea while climbing stairs and some chest tightness with exertion. (CX 1-7). Among other factors, Dr. Childs noted that Claimant "actually had a severe obstructive defect and a mild restrictive defect with the mild reduction in his TLC," and [t]here was minimal biapical pleural thickening." (CX 3C-A). Dr. Childs' initial impression was that Claimant had severe chronic obstructive pulmonary impairment, but he could not tell if it was a significant component because no bronchodilator was given. (CX 1-8). He noted that he was concerned about Claimant's abnormal cardiology work-up and stated "[t]hese [cardiac] changes would make me concerned that the patient's obstructive defect might be fixed." Dr. Childs adjusted Claimant's medications, advised him that he would need another full PFT work-up and referred him to a cardiologist. (EX 1-8). *See also* (CX3C-A),(CX3D-A), (EX 12-6, 12-7).

Dr. Derrick Ridley examined Claimant on August 3, 1994. During the examination, Dr. Ridley reviewed Claimant's history of present illness, his past medical history, his allergies and medications, his family and social history, the results of his own physical examination, EKG, and echocardiogram results from June 1, 1994. After considering all of this information, Dr. Derrick Ridley makes the following assessments:

(1) Chest pain of unspecified etiology. The patient's chest pains are oppressive in nature and inconsistently associated with exertion. Rule out significant atherosclerotic coronary artery disease and coronary insufficiency.

(2) Mild right ventricular enlargement most likely related to the patient's lung disease.
(3) Asthma
(4) Rule out Alpha I anti-trypsin deficiency.
(5) Allergy to Iodine.

(CX 4-8). Dr. Ridley then recommends a Thallium-Exercise Stress test, Alpha I anti-trypsin level, and a thyroid profile. *See generally* (CX 4-6 thru 4-8), (EX 14-21, 14-22).

In a pulmonary function report dated August 5, 1994, Dr. Childs approved the interpretation and impression noted at the pulmonary function laboratory. That impression was:

> Combined mild obstructive/restrictive defect with hyperinflation. There is significant improvement in both the FVC and FEV_1 since the previous study.

(CX 3E-A). *See also* (CX 3F-A, 3G-A, 3H-A, 3I-A)(results of Claimant's study).

In a note dated August 8, 1994, Dr. Ridley stated the results of Claimant's Nuclear Medicine Stress and Resting Spect Thallium Myocardium Imaging Study. (EX 14-8). Dr. Ridley noted that "an excellent level of exercise was achieved with the patient reaching 94 % of the predicted heart rate." *Id.* His impression was: "[t]here is mild fixed thinning of the inferior wall suggesting scar. Minimal reversible ischemia is noted in the septum." (EX 10-8). The radiologist was Stephen A. Fink.

In a letter to Dr. Acosta dated August 17, 1994, Dr. Derrick E. Ridley writes describing his examination of Claimant on August 3, 1994. Dr. Ridley was asked to consult to assist in the evaluation of Claimant's chest pain and dyspnea. Dr. Ridley notes that his impression was that Claimant's chest pains were atypical for coronary insufficiency. He supervised Claimant's stress test and found him to have "excellent exercise tolerance without experiencing chest pain during this treadmill." He also noted that there were no cardiac dysrhythmias during the exercise and that it was normal by the electrocardiographic criteria. (CX 4-4). Dr. Ridley states that it is his impression that Claimant does not suffer from significant heart disease. (CX 4-5). He states that if symptoms continue to be unexplained, a coronary angiography should be performed. *Id.* The notes from his examination of Claimant, from which he drew the above conclusions, is in evidence at CX 4-6, 4-7. *See also* (CX 4-10)(results of test, dated February 17, 1999).

In Dr. Childs' notes, dated September 6, 1994, he uses a shorthand notation system of S.O.A.P. which has not been explained in the record. Dr. Childs did clearly state that he examined Claimant on a follow-up of chronic obstructive lung disease and possible mild reflux esophogitis. (CX 1-6.) A cardiology evaluation by Dr. Derrick Ridley did not show any significant coronary artery disease. (CX 1-6.) Under "A:", Dr. Childs indicated a "[M]ild COPD which markedly improved the Claimant's initial complaint of SOB." He indicated under the heading "P:" that no further pulmonary evaluation was needed. (CX 1-6.) *See also* (CX 3A-A), (EX 12-4).

Office note of Dr. Acosta dated October 5, 1994, again uses S.O.A.P. shorthand. The

note is mostly illegible, however, under A. "COPD" is noted. (EX 17-31).

Office note of Dr. Acosta dated November 7, 1994, again uses S.O.A.P. shorthand. The note is mostly illegible, however, under A. "COPD" is noted. (EX 17-30).

Office note of Dr. Acosta dated November 16, 1994, again uses S.O.A.P. shorthand. The note is mostly illegible, however, under A. "COPD" is noted. (EX 17-29).

Dr. James V. Scutero performed an "Asbestos Evaluation" of the Claimant on February 28, 1995. He noted that the Claimant had worked for Newport News Shipbuilding as a welder since 1968, and that he had been exposed to asbestos insulation "almost on a daily basis from 1968-1980 at which time protective devices were put into play." In addition to his physical examination, Dr. Scutero performed a chest x-ray and pulmonary function study. Dr. Scutero stated that the x-ray was within normal limits and that the pulmonary function studies were "normal except for a mildly decreased vital capacity." He diagnosed that the Claimant had no evidence of asbestosis, but that he should be checked periodically because of his history of contact with asbestos. (CX 1A-A). The laboratory reports, dated February 27, 1995, accompany Dr. Scutero's report. *See* (CX 1B-A); (CX 1C-A). The report confirms Dr. Scutero's impressions. It says: "Normal flows; Vital capacity is reduced; Normal flow volume loop; Normal diffusion capacity; Normal study except decreased vital capacity." (CX 1B-A).

In Employer's Clinic records it is noted that on March 5, 1995, "record made for asbestos related lung disease." (EX 10-41).

In a report of occupational illness form dated March 5, 1995, Claimant states that he was injured by "inhalation of airborne asbestos dust and fibers." It is noted on this form that the reporting date was February 20, 1995, the injury date is noted to be June 24, 1994, and it is noted that it is controverted as not arising out of or in the course of employment. The illness is noted as asbestos related lung disease and the name of the doctor is Dr. George C. Childs, Jr. (EX 10-54).

In a note dated February 27, 1995, on Dr. Scutero's letterhead, signed by Leo P. O'Connell, M.D., a report on Claimant was issued. It stated the date of the report was March 13, 1995. The report gave "R/O Pulmonary Fibrosis" as Claimant's diagnosis. It also stated that Claimant's lateral chest x-ray showed "[c]ardiac enlargement without evidence failure. Scarring.... No interstitial fibrosis seen." It is noted that the copy provided the Court is cut off on one side and therefore can not be fully read. Finally, the report indicated that Claimant suffers "[c]ardiac enlargement without evidence failure." (CX1D-A).

In a report dated January 20, 1998, Dr. Ray A. Harron apparently reviewed Claimant's chest x-rays dated November 13, 1997. His impressions of those x-rays is that they are "consistent with asbestosis." Dr. Harron is Board Certified in Radiology and is a NIOSH B reader. (EX 23).

The office note of Dr. Acosta dated March 18, 1998 uses the S.O.A.P.shorthand, but the note is illegible. (EX 17-13).

The office note of Dr. Acosta dated May 5, 1998 uses the S.O.A.P.shorthand. It appears that this visit concerned chest pain. (EX 17-13).

In a letter to Claimant's counsel [in the asbestos civil litigation] Mr. Nicholl, dated October 9, 1998, Dr. Steven M. Zimmet reported on his examination of Claimant on September 28, 1998. (EX 22). On that day Claimant had complete pulmonary function tests in addition to a history and physical examination. Dr. Zimmet also reviewed Claimant's chest x-rays. Under the heading "Data" Dr. Zimmet noted that Claimant's November 13, 1997 x-ray shows "increased pulmonary parenchymal markings indicative of bilateral interstitial pulmonary fibrosis. These films were also reviewed by Ray A. Harron, M.D. who agrees they are consistent with asbestosis." Dr. Zimmet also opined:

> Pulmonary function tests demonstrate a mild decrease in flow and vital capacity with a mild increase in residual volume and a moderate decrease in diffusing capacity. These test results are indicative of a mild obstructive lung defect with a mild reduction in vital capacity and a moderate reduction in gas exchange.

(EX 22-2). Based upon Claimant's occupational history of asbestos exposure, abnormal pulmonary function tests and abnormal chest x-rays, Dr. Zimmet's assessment is that Claimant suffers from asbestosis. (EX 22-2).

Office notes of Dr. Acosta, dated November 10, 1998. In this note Dr. Acosta uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. (CX 3-10), (EX 17-10).

Office notes of Dr. Acosta, dated November 18, 1998. In this note Dr. Acosta uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. It is clear that he writes "send letter to shipyard indicating pt. on restriction due to breathing problems." The remainder of the note is illegible. (CX 3-10), (EX 17-10).

A letter from Dr. Carlos Acosta, dated November 19, 1998, indicates that Claimant was thoroughly evaluated for shortness of breath in 1994 by a cardiologist and a pulmonary specialist. (CX 3-1), (CX 3-16), (EX 17-1). Dr. Acosta states that the final evaluation was that Claimant has severe chronic obstructive pulmonary disease with minimal improvement with bronchodilators. *Id*. Dr. Acosta stated that since that time, Claimant has become sensitive to smoke, irritant inhalants and even using a respirator causes Claimant to have shortness of breath. Dr. Acosta, therefore, opined that Claimant would benefit from better environmental conditions at work, specifically in areas that do not require a respirator. *Id*.

Office notes of Dr. Acosta, dated December 2, 1998. In this note Dr. Acosta uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. Dr. Acosta writes "A: COPD." (CX 3-11), (EX 17-9).

Office notes of Dr. Acosta, dated December 7, 1998. In this note Dr. Acosta uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. (CX 3-11), (EX 17-9). It is clear in a separate notation that Dr. Acosta referred Claimant to Dr. Childs on this date.

In a report dated December 8, 1998, interpreting Claimant's chest x-ray of December 7, 1998, radiologist Kishore Rao writes:

> Lungs remain clear of acute infiltrate. Minimal parenchymal scarring is unchanged. Heart size is at the upper limits of normal. Left ventricular contour is slightly prominent.
>
> Conclusion: Radiographic appearance is unchanged as compared with previous examination of January of 1994. No acute pulmonary infiltrates are identified.

(CX 3-26), (CX 7-6), (EX 9-2, 9-3).

Office notes of Dr. Acosta, dated December 9, 1998. It is noted here that labs were drawn. (CX 3-11), (EX 17-9).

Office notes of Dr. Acosta, dated December 16, 1998. In this note Dr. Acosta uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. (CX 3-12), (EX 17-8).

In a letter to Dr. Acosta, dated December 18, 1998, Dr. Childs indicates that he believed the dyspnea was related to Claimant's improper use of his Serevent MDI. (CX 1-5). Serevent is a BID drug which lasts 12 hours, it cannot be used on a PRN basis. *Id*. Claimant demonstrated the technique he had been using to administer the MDI, which was wrong. It is designed to exhale first, then administer while inhaling to total lung capacity. Claimant had been inhaling to TLC, the administering drug on exhale. Dr. Childs said that Claimant's long history of reactive airways diseases (asthma) can have restrictive defects. *Id*. Claimant was instructed on how to use his medication, and was scheduled for more pulmonary tests. *Id*. *See also* (EX 12-2, 12-3).

The office note of Dr. Acosta, dated December 18, 1998 is largely illegible, but appears to note a call. (CX 3-12), (EX 17-8).

On a form dated December 22, 1998, Dr. Acosta noted that Claimant was being seen for shortness of breath and a cough. He was first seen for this disability on November 10, 1998. He wrote that it was unknown if this disability arose out of employment. He explained Claimant's condition: "This patient has lung changes [difficult to read] of chronic obstruction. What caused it 'I don't know.' Pt. has been referred to a lung specialist." He also noted that Claimant should be restricted at work by the lung specialist. In the specific treatment plan for Claimant he noted "avoidance of pollutants to respiratory system." (CX 3-2). He further noted that Claimant was continuously disabled from November 10, 1998 to December 16, 1998. *See also* (EX 11-1, 11-2).

The office notes of Dr. Acosta, dated December 28, 1998, note a referral but the doctor's name illegible. (CX 3-12), (EX 17-8).

The interpretation of a pulmonary function test performed at Sentara Hampton General Hospital on December 29, 1998 (CX 3-22), indicates a moderate reduction in the FVC and FEV_1 with a normal FEV 1%, moderate reduction in the FEF 25-75, and no significant improvement post-bronchodilator; a moderate reduction in the MVV, a mild reduction in the TLC and FRC; a normal RV; normal DLCO; an essentially normal flow loop, except for terminal slowing of the expiratory limb, and a normal ABG. (CX 3-23.) The impressions were, when compared to a previous study performed on 8/5/98, that Claimant's FVC, FEV_1, and TLC was essentially unchanged. Claimant has a combined mild restriction with a small airways dysfunction. The interpreter suggested clinical correlation. (*Id*.) (CX 7-7), (EX 5-2).

Dr. Childs' office notes on December 30, 1998, indicate that Claimant was in his office for a follow up after an evaluation on December 18, 1998 for dyspnea. Again, Dr. Childs used the S.O.A.P.shorthand that has not been explained. At that time, Claimant had stated that he had "been off his pulmonary medications until January 1998." Claimant then reported that he restarted taking his Serevent as needed. Claimant was complaining of chest tightness at night and reported that he had not been to work because of his shortness of breath. (CX 1-2).

Dr. Childs noted that Claimant's physical examination revealed "lungs to be clear to auscultation and percussion." (CX 1-2). Dr. Childs also noted that Claimant continued to complain of dyspnea with minimal exertion. He stated that Claimant's dyspnea did not appear to be on the basis of a significant pulmonary impairment. (CX 1-3). He also stated that Claimant did have a mild restriction on pulmonary function tests, but he believed that part of the defect was caused by Claimant's body habitus. *Id*. Claimant is underweight and the pulmonary function tests are based on a person of average height and weight. Moreover, when his pulmonary function test values were reviewed against the values outlined by the AMA guidelines, claimant fell between a Class 1 and a Class 2 impairment (which means between no impairment and a mild impairment of the whole person.) Dr. Childs stated that exercise induced hypoxemia rarely occurs when the DLCO is greater than 60% of predicted. (CX 1-3). Dr. Childs scheduled Claimant for a pulmonary exercise study because his dyspnea is out of proportion to his clinical and psychological findings. (CX 1-3). Finally, Dr. Childs noted that Claimant inquired about going back to work and he informed him "I do not have a pulmonary indication at this point to recommend that he not return to his duties." (CX 1-3).

In a note that appears to be dated January 4, 1999, an illegible note in Claimant's chart at Employer's Clinic says "to Dr. McCune" and appears to state "breathing problems." Again the S. shorthand is used, which has not been explained as subjective or objective symptoms. (EX 10-43).

In a form entitled Report of Occupational illness, dated January 4, 1999, Claimant reported "I have been having breathing problems. I feel it is related to welding over the years." Under diagnosis it is written "COPD of unknown etiology." (EX 10-3). Also, in Employer's Clinic Administration System, it is noted that Claimant has a permanent restriction of "no use respirator or exposure to dusty environments." (EX 10-4).

On a form dated January 5, 1999, Dr. Acosta listed Claimant's disabilities as COPD and fatigue. His first date of treatment for this disability was noted as December 16, 1998. Dr. Acosta indicated that this disability did not arise out of patient's employment but that it "is aggravated by fumes." He noted that Claimant was continuously disabled from December 16, 1998 to January 4, 1999 (month unclear). (CX 3-3). *See also* (EX 11-3, 11-4). The Notice of Controversion was also filed on this date. Occupational disease of respiratory system was listed. (EX 3).

Employer's first report of injury, dated January 6, 1999. This report indicates that Claimant suffers a respiratory system, occupational disease. The first treating physician was chosen by the employee and the date of authorization is November 18, 1998. Claimant reported "I have been having breathing problems, I feel it is related to welding over the years." (CX 9), (EX 1).

A pulmonary function test performed by Dr. Jeffery Scott, dated January 7, 1999, indicates that Claimant's resting pulmonary function revealed a forced vital capacity of 3.54. Liters or 66 % of predicted. His FEV_1 was 2.92 liters or 67% of predicted, and his FEV_1/FVC ratio was normal. These values suggest a restrictive ventilatory defect. (CX 7-1). Dr. Scott's interpretation was that it was a maximal exercise study, as the patient achieved 86% of his predicted heart rate. (CX 7-2). Dr. Scott noted that Claimant had a hypertensive response to exercise and a decreased maximum oxygen consumption from predicted value. (CX 7-2). The normal breathing reserve, early anaerobic threshold preserved oxygenation and hypertensive response with exercise suggests a possible cardiovascular etiology to exercise limitation. *Id.* No electrocardiogram changes suggestive of ischemia were noted during exercise or recovery. (CX 7-2). Dr. Scott noted that serial testing might be helpful in determining the source of exercise limitation in Claimant. (CX 7-2). *See also* (CX 7-9), (EX 7-2, 7-3, 7-5).

Dr. George G. Childs, Jr.'s office notes from January 29, 1999 indicate that Claimant was in his office for a follow-up from a previous visit. Dr. Childs indicated that there was no significant change in Claimant's pulmonary function tests. Dr. Childs stated that Claimant did have a moderate restrictive defect, but that the Claimant's DLCO was normal. Claimant was sent for a pulmonary exercise study on 1/7/99 because of his complaints. (CX 1-1.) At that time, Claimant was noted to achieve 86% of his predicted heart rate, although he was noted to have a hypertensive response to exercise. *Id*. Claimant's maximum oxygen consumption was decreased and he has a normal breathing reserve, but an early anaerobic threshold. Dr. Childs stated that it was the feeling of the interpreter that the findings suggested a possible cardiovascular etiology to Claimant's exercise limitations. *Id*. Dr. Childs also commented that Claimant had an abnormal pulmonary exercise test which suggested a cardiovascular limitation to exercise. He questioned whether this patient had a Marfanoid-type Syndrom with cardiovascular involvement. Dr. Childs recommended a reevaluation by cardiologist since there was no evidence of restriction with desaturation which would suggest significant debilitating lung disease. *Id*. *See also* (EX 12-1).

The office note of Dr. Acosta, dated February 1, 1999 uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. (CX 3-13).

The office note of Dr. Acosta, dated February 2, 1999, notes a referral to Dr. Ridley. (CX 3-13), (EX 17-7).

In a note dated February 4, 1999 it is noted that Claimant has test set up for February 5, 1999, and a cardiac work up. It is also noted that he will see a pulmonologist, Dr. Shaw on that date. (EX 10-43).

In Employers Clinic Administration system, dated February 5, 1999, it is noted again that Claimant has permanent restrictions that say "No use respirator or exposure to dusty environments not to work in a high fume exposure environment." (EX 10-5). In what appears to be a copy of this form, updated on February 25, 1999, it states "[a]t this time this injury is personal see SEQ 001...NNS Dr. McCune/RAH." (EX 10-6).

The office notes of Dr. James O. Shaw, dated February 5, 1999, indicate that Claimant was being evaluated as per the request of Employer. He noted that upon physical examination, Claimant's chest was "clear without wheezes" and that there was "no wheeze on forces expiration." (EX 13). Dr. Shaw found that the pulmonary function tests showed borderline restrictive defects with an FVC of 73%, but within 95 % confidence levels of normal. (EX 13-2). Claimant's TLC was 84 %, which Dr. Shaw thought was normal, and Claimant's DLCO was normal at 140 %. Dr. Shaw also noted that Claimant showed a 50% improvement in FEF post-bronchodilator. (EX 14-2). Dr. Shaw thought Claimant's chest x-ray showed a slightly narrowed AP diameter although he did not see definite pectus excavatus. He also noted that the lung fields and pleural stripes were clear. (EX 14-2).

Dr. Shaw's impressions were: "[d]yspnea and shortness of breath of unknown etiology, at least partially due to underlying asthmatic defect with a marked response to bronchodilators. Probably some exacerbation of underlying asthmatic symptoms by exposure to welding fumes." (EX 14-2).

Dr. Shaw went on to say that he felt that Claimant should not work in a high fume exposure environment and that because of this underlying asthma, he did not think he could wear a respirator effectively. (EX 14-2). Dr. Shaw's Curriculum Vitae is in evidence at EX 19.

In a form dated February 9, 1999, Claimant filed a report on his injury. He listed as his disability "severe chronic obstructive pulmonary disease." Again, this was filed by Claimant, not a doctor. (CX 11), (EX 2).

In a note describing his consultation with Dr. Acosta regarding Claimant's heart condition, Dr. Derrick E. Ridley noted that Claimant's lungs were clear and that his heart had a regular rate and rhythm. His impressions were chest discomfort and dyspnea of uncertain etiology; asthma; and allergy to iodine. His recommendations were to have a Thallium exercise stress test and echocardiogram performed and, if not performed recently, a complete metabolic profile and CBC. (CX 4-2, 4-3), (EX 14-6, 14-7).

The office note of Dr. Acosta dated February 14, 1999, acknowledges that he received a note from Dr. Childs regarding Claimant's pulmonary exercise test. He writes:

> Note Dr. Childs: Abnl. [Abnormal] pulmonary exercise test suggesting a cardiovascular limitation to exercise. Pt. should have re-evaluation by cardiology since he does not have evidence of restriction c desaturation which would suggest significant debilitating lung disease.

(CX 3-13), (EX 17-7).

The office note of Dr. Acosta, dated February 16, 1999, again uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. Additionally, the copy quality is poor, rendering it largely illegible. (CX 3-13), (EX 17-7).

The results of Claimant's Spect Thallium Stress Test were interpreted by Dr. Michael Yedinak. His impressions were that Claimant had a good exercise tolerance, no chest pain or dysrhythmias, normal exercise stress test and a normal thallium stress test. (CX 4-1), (EX 14-17).

The office note of Dr. Acosta, dated February 22, 1999, uses the shorthand S, O, A, P. This shorthand makes it impossible, without explanation, to ascertain what the doctor is observing and what are subjective complaints of the Claimant. (CX 3-14). (EX 17-6).

In a note dated February 24, 1999, Dr. Ridley reviews the results of Claimant's Thallium stress test results, noting a good exercise tolerance, and his unremarkable echocardiogram results. His assessment is: "Stable cardiac condition. It is quite doubtful that the patient's chest pain and dyspnea are due to ischemic heart disease." (EX 14-12).

In a note on Claimant's chart with Employer's clinic, again using the S.O.A.P. shorthand, it is noted under A "non-Occ at this time." Claimant's restrictions are also noted "[n]o use –pressure respirator. No dust, irritant fume environment." (EX 10-46).

The office note of Dr. Acosta dated February 26, 1999, seems to note a cardiology evaluation. The note is short and largely illegible. (CX 3-14), (EX 17-6).

In a letter to Employer's counsel, dated March 18, 1999, Dr. Shaw stated that he had reviewed Claimant's medical records which included his examination of Claimant on February 5, 1999, the evaluations of Dr. Ridley. (EX 14-3). Dr. Shaw's opinion was that Claimant had a history of bronchial asthma and on Dr . Shaw's evaluation, showed a positive bronchodilator response indicating reactive airways disease or bronchial asthma. Dr. Shaw felt that welding fumes probably exacerbated this underlying condition. (EX 14-3). Dr. Shaw opined:

> [t]he exacerbation by the welding fumes more than likely to a reasonable degree of medical certainty was temporary in nature and would resolve withing a relatively short period of time after being out of the exposure. Moreover, welding fumes did not cause the asthma and should not cause any permanent worsening of his asthmatic symptoms once removed from the exposure.

(EX 14-3).

The office note of Dr. Acosta dated March 22, 1999, uses the S.O.A.P.shorthand. It appears to be a visit regarding shortness of breath. (EX 17-5).

In an essentially illegible report, a report from a doctor of Employer's clinic, whose name appears to be Dr. McCune, dated April 2, 1999, all that is clear are the words "of unknown etiology." (CX 8-1). The Clinics administrative reports are in evidence at CX 8-2, 8-3, 8-4).

The office note of Dr. Acosta dated April 12, 1999, refers Claimant to Dr. Baker. (EX 17-4).

Dr. James P. Baker examined the Claimant on April 14, 1999. In this new patient evaluation Dr. Baker relates Claimant's symptoms as well as his employment and medical history. Dr. Baker noted that, probably during the first ten years of his employment, Claimant was exposed to "significant asbestos...as he was using asbestos to protect himself from welding burning material as well as being exposed to asbestos being removed from various pipes and other things that he was having to weld." According to Claimant's report, his current problem began around the 1980's, when he began to have "some shortness of breath but really the majority of his problem began around 1994 at which time he noted significant exertional dyspnea." At this time he was told that "he did not have significant cardiovascular disease and that he had some restrictive and obstructive airways abnormality and was placed on bronchodilator medication." Claimant stated that he is currently unable to work due to his shortness of breath when climbing stairs and other physical exertions. Dr. Baker noted that "[Claimant] is continuing to have significant difficulty and to be remarkably limited in his ability to do things from dyspnea...." Claimant was not, at this time: coughing; producing any sputum; having any significant acute respiratory problems; losing weight or have any significant heat intolerance.

After a physical examination, Dr. Baker ordered a chest x-ray and set out to obtain the results of Claimant's cardiopulmonary exercise test and pulmonary function studies. Although Dr. Baker writes that he will complete this dictation after reviewing these materials, there is no further information available as to this date. (CX 2A-A; 2B-A), (CX 5-10, 11), (EX 15-9, 15-10).

In a report dated April 15, 1999 from Virginia Beach General Hospital, Radiology Consultation and approved by Felix A. Hughes, M.D., Claimant underwent two chest x-rays at the request of Dr. Baker. The diagnosis on this report states:

> Two views of the chest are presented with no prior study available for comparison. There is mild prominence of the ascending aorta but I see no acute infiltrates or failure. The heart and mediastinal structures are within normal limits. There is mild trivial rotoscoliosis of the dorsal spine.

(CX 2C-A). The impression was "[n]o acute cardiopulmonary abnormality seen." *Id.* *See also* (CX 5-12) (CX 7-5)(EX 9-1).

In a letter to Dr. Acosta from Dr. Baker, dated May 7, 1999, Dr. Baker reports on his evaluation of Claimant for asbestos related disease. Dr. Baker writes:

> I have reviewed [Claimant's] chest x-ray. He has a question of a slight increase in irregular densities scattered throughout his lower lung fields though it is certainly not of a level of significance. His B reading is profusion of 0/1. There are no pleural plaques.
>
> I have indicated to [Claimant] that I do not think he has asbestosis. He clearly has asbestos exposure. He has an abnormality in his pulmonary function which is compatible with asbestosis but his chest x-ray does not indicate pulmonary asbestosis and he does not have any crackles in his chest. ...
>
> At the current moment I do not know exactly what the etiology of [Claimant's] exertional dyspnea is. I am concerned about the fact that he has a narrow AP chest diameter which might be creating some problems from a cardiac standpoint and the factor that he does have slight restriction on his pulmonary function studies.

(CX 2D-A, 2E-A). Finally Dr. Baker states that, as of this date, he is uncertain of the etiology of Claimant's slight restriction. (CX 2E-A). (CX 5-8, 5-9).

In a letter to Dr. Acosta dated May 7, 1999, Dr. Baker indicated that Claimant's pulmonary function tests demonstrate both a mild obstructive and a restrictive abnormality with normal diffusion capacity. (CX 5-8). Dr. Baker opined that he did not believe Claimant had asbestosis, but that he clearly had asbestos exposure. (*Id*.) He thought Claimant had an abnormality in his pulmonary function which is compatible with asbestosis, but that his chest x-ray does not indicate pulmonary asbestosis and he does not have any crackles in his chest. (CX 5-8). Dr. Baker went on to say that he does not know exactly what the etiology of Claimant's exertional dyspnea is, but he thinks it might be the fact that Claimant has a narrow AP chest diameter which might be creating some problems from a cardiac standpoint and the factor that he does have slight restriction in his pulmonary studies. At this point the etiology of that was uncertain in Dr. Baker's mind. (CX 5-9), (EX 15-7, 15-8).

The office note of Dr. Acosta dated May 26, 1999, uses the S.O.A.P. shorthand. It appears to be a visit regarding shortness of breath. (EX 17-4).

In a letter to Dr. Acosta dated June 2, 1999, Dr. Baker states that, after further research, it occurred to him that Claimant may have "diastolic dysfunction cardiac wise." Dr. Baker encourages Dr. Acosta to explore that possibility. (CX 2F-A), (CX 5-7), (EX 15-6).

In an office note dated June 4, 1999, Dr. Derrick E. Ridley noted Claimant's "problem list" as chest pain of uncertain etiology, dyspnea on exertion, and history of asthma. He notes that Claimant denied having chest pain, tightness or fullness. He noted that Claimant's chest was clear. His assessment was "[s]table cardiac condition." (EX 14-1).

In an office note dated June 16, 1999, Dr. Ridley notes his assessment of Claimant as "[u]nexplained chest pain and dyspnea." He also notes that Claimant is allergic to iodine. His plan is to proceed with a right and left heart catheterization with coronary and left ventricular

angiography. (EX 14-9).

Dr. Ridley noted normal, unremarkable results an echocardiogram report dated June 22, 1999. (EX 14-19).

In a note dictated June 22, 1999, Dr. Ridley recorded Claimant's social and medical history. This note is apparently a part of Claimant's admittance into the hospital for a diagnostic cardiac catheterization. (EX 14-2, 14-3).

In a note dictated June 26, 1999, Dr. Ridley recorded the results of Claimant's heart catheterization and concluded all was normal except for "significant single vessel atherosclerotic coronary artery disease involving the non-dominant right coronary artery." (EX 14-15, 14-16).

In a note dated July 7, 1999, Dr. Ridley noted that Claimant had undergone a cardiac catheterization on June 25, 1999 and that he was found to have "significant single vessel atherosclerotic coronary artery disease involving the nondominant right coronary artery. All other results appear to be normal. His assessment is:

> 1. Significant atherosclerotic coronary artery disease involving a small nondominant right coronary artery. There is no evidence for left ventricular or right ventricular diastolic dysfunction.
>
> 2. Unexplained dyspnea.

(EX 14-4). He noted that he advised Claimant to have a Lipid profile performed in the near future and to take on aspirin per day. (EX 14-5).

In a physical capacities evaluation dated July 12, 1999, Dr. Acosta restricted Claimant from exposure to fumes, dust, and chemicals. He wrote that Claimant was permanently disabled for his type of work from November 18, 1998. There is evidence of frequent dyspnea and chest pain. He expects Claimant's condition to deteriorate with age and continuous exposure. (CX 3-4 thru 3-7). *See also* (EX 17-51 thru 17-53)(Dr. Acosta's physical capacities evaluation, dated July 12, 1999 that essentially says see specialist's report). It should be noted that in EX 17, Dr. Acosta's note that he expects Claimant's condition to deteriorate is found in Employer's form, not the physical capacities form.

Also on July 12, 1999, Dr. Acosta (apparently, signature illegible), also completed a disability retirement claim for Employer. In this form he indicated that Claimant's symptoms were "shortness of breath, especially on exertion and when exposed to fumes dust and humidity." Claimant was noted to have reduced breath sounds. Claimant's right coronary artery condition was noted to be a secondary condition contributing to the disability, although he has no limitation due to this condition. The symptoms first appeared in 1994 and he was first unable to work on November 18, 1998. Dr. Acosta noted that this condition is work related due to exposure to welding fumes and dust at work. His primary diagnosis is COPD. (CX 3-8), (EX 17-49, 17-50). Dr. Acosta notes that specialists will forward their reports individually. This same form is in evidence at CX 5-1, in Dr. Baker's records, as well.

In a form from Employer entitled Disability Retirement Claim-Physician's statement, dated July 28, 1999, Dr. Baker indicates that Claimant's primary diagnosis is restrictive lung disease. Dr. Baker notes that Claimant's symptom is exertional dyspnea, and that his objective findings are "narrow AP chest diameter, decreased chest expansion and -[illegible word]-lung volumes." He states that there are no secondary conditions contributing to the disability and that the symptoms first appeared in 1994 and Claimant was first unable to work on November 18, 1998. He writes that Claimant's condition is work-related "Pt. has worked in paint and chemical [illegible words] & has developed restrictive lung abnormality and a minimal obstructive abnormality." He also noted that he referred Claimant for a cardiology consultation. (CX 5-2). Dr. Baker wrote that Claimant cannot perform tasks which require manual labor. There is another restriction however it is illegible. (CX 5-3).

On a separate physical capacities evaluation dated July 28, 1999, Dr. Baker restricts Claimant to 1-2 hours of standing or walking in a work situation allowing for periodic breaks to rest in a 7-8 hour day. He states that Claimant could not stand or walk as long as 2 hours without an opportunity to sit. He further states that Claimant could sit for 5 to 6 hours in a sitting job where he could shift positions as necessary and occasionally stand. Claimant could work for a full 8 hours if he could change from sitting to standing at will.

Dr. Baker also restricted Claimant to occasional lifting and carrying of weights between 0 and 10 pounds and restricted him completely from lifting and carrying weights from 10 to 50 pounds. He noted that there was evidence of frequent dyspnea with a degree of severity with light exercise. There was also evidence of chest pain. Finally Dr. Baker noted that it is medically necessary that Claimant lie down or sit to rest or be inactive for a certain period during an 8 hour work day. (CX 5-5, 5-6).

In office notes dated July 28, 1999 Dr. Baker documents his examination of Claimant. Dr. Baker uses the shorthand of S. and O. that has not been explained to his court. He clearly states, however, that Claimant states that he is "reasonably okay if he is in a cool air conditioned area but if he is outside where it is warm he has difficulty if he has to do any significant exertion he becomes rather short of breath." Claimant last worked in early November of 1998, prior to which he had gradually progressive respiratory difficulty for approximately 4-5 years. As best Claimant and his wife can remember, there is a specific incident, the painting of a ship with some form of unusual paint, after which "he has continued to have significant problems somewhat more than he ever had before and that has gradually lead to the problem that he has at the ...moment." (CX 2G-A).

During his examination of Claimant Dr. Baker noted: "Chest reveals a straight dorsal spine, a narrow AP diameter and significant decrease in excursion of his chest wall. Breath sounds are somewhat diminished. I do not hear any crackles or wheezes today." (CX 2G-A). In attempting to reach a diagnosis for Claimant, Dr. Baker writes:

> Dr. Ridley has effectively ruled out the possibility of diastolic dysfunction in his heart as a cause for his dyspnea thus we are left with a pulmonary parenchymal abnormality or a chest wall abnormality. The presence of a normal diffusion capacity on his pulmonary function studies suggest that the problem is not

> pulmonary parenchymal abnormality. [Claimant] does have a significant restrictive abnormality with some also probably small airway obstruction abnormality. ...
>
> [Claimant's] dyspnea is not completely explained in my mind nor is his restrictive lung disease. He did not have difficulty prior to working in his current occupation. He is never smoker an does not have hobbies which put him into any type of inhalant irritants. I think [Claimant] must be kept in a work environment which is clean, air conditioned and he should not be requested to return to a position in which he is expected to do any significant physical labor or be exposed to chemicals, dust, dirt, smoke, etc.

(CX 2H-A). See also (EX 15-1, 15-2).

In a memo received on September 2, 1999, Dr. Joseph Ross stated that he reviewed Claimant's medical records, including the records of Employers Clinic; chest roentengrams on 11/14/68, 3/2/71, 7/17/74, 3/23/76, 4/2/76 and 4/15/99; pulmonary function studies dated 5/13/94, 8/5/94, 12/29/98, 1/7/99; an echocardiogram dated 6/1/94; and the medical records of Dr. Childs, Dr. Acosta, Dr. Shaw, and Dr. Baker.

Dr. Ross's impressions were that there was no evidence that Claimant has a work-induced disease, but considerable evidence against it. (EX 16.8.) Dr. Ross indicated that there is no roentgenographic evidence of asbestos exposure, therefore, Claimant did not have pulmonary asbestosis. Dr. Ross stated that the chest films do not show evidence of siderosis, the lung condition commonly associated with welding fume exposure. Dr. Ross also stated that Claimant did not have bronchitis, which is associated with exposure to dust and fumes, since Claimant's cough, wheezing and sputum products had not been significant components of his illness (EX 16.9.)

Dr. Ross stated that the pulmonary impairment has frequently been classified as a combination of obstructive and restrictive processes. The pulmonary function test performed on 5/13/94 pointed to an obstructive process with reductions in FVC, FEV_1/FVC, MMEF, and MVV and above normal FRC and RV. (EX 16.9.) Dr. Ross states that in restrictive lung disease, all lung volumes are equally reduced, except possibly in chest wall or muscle weakness, in which case residual volumes may be normal and expiratory volumes and flow rates are reduced, resulting in a low measured TLC. Dr. Ross states that another problem he sees in the results of the pulmonary function test was that no correction was made for African Americans who have a lower spirometery values than north American White. After making this correction to the pulmonary function test done on 1/7/99, Claimant's FVC predicted normal would be 4.44L and the measured 3.54L would be 80% of normal. The FEV_1 predicted normal would be 3.81L and the measured value of 2.92 would be 77% of normal. Dr. Ross stated that the values then were borderline normal. (EX 16.9.)

Dr. Ross opined that, in view of the cardiac evaluation, it seemed most likely that Claimant's dyspnea was related to a relatively mild pulmonary impairment. (EX 16.9.) Dr. Ross stated that Claimant did not have a restrictive lung disease such as asbestosis and he did not have chronic bronchitis. Dr. Ross thought he might have small airways disease without wheezing as an

adult manifestation of childhood asthma. He thought Claimant could also have a chest wall impairment or muscle weakness which could account for his problem. (EX 16.10.) Dr. Ross stated that the only effect of dust and fumes would be to accentuate his symptoms temporarily, but that he agreed that the basic underlying impairment was neither caused by nor made worse in any significant way by his work environment. (EX 16.10.). *See also* (EX 18)(Dr. Ross' Curriculum Vitae).

Dr. James P. Baker gave a deposition on November 2, 1999. That deposition was entered into evidence as CX 6. Dr. Baker's Curriculum Vitae was also entered into evidence, attached as Deposition Exhibit #1. (CX 6-40 thru 6-56). *See also* (CX 6-29). Dr. Baker is a specialist in pulmonary medicine and Board Certified in Internal Medicine, but there is no indication that Dr. Baker is Board Certified in Radiology or that he is a NIOSH B reader (CX 6-3). He first saw Claimant on April 15, 1999. *Id.*

Asthma

Although Claimant did tell Dr. Baker's secretary that he had a history of childhood asthma, it was not in Dr. Baker's initial report. (CX 6-4). Dr. Baker testified that he did not consider Claimant's childhood asthma as significant because Claimant had indicated that "he did not have any difficulty in early adulthood from the time he finished high school until he told [Dr. Baker] that he began having problems in the late 1980s." (CX 6-4, 6-5). According to Claimant's description of his problems to Dr. Baker, he began having shortness of breath around the late 1980s. (CX 6-5). Dr. Baker testified that as he reviewed Claimant's records from Dr. Childs, he saw that Claimant began seeing Dr. Childs in the earlier 1980s. *Id.*

Dr. Baker testified that he knew Claimant had childhood asthma that resolved in his adult years, per Dr. Childs' records from the mid 80's. He testified that he did not know that Claimant had undergone desensitizations. He knew that Claimant had a history of using inhalers during his adult life. (CX 6-19, 20).

1994 Pulmonary Function Test

Dr. Baker stated that, according to Dr. Childs records, at that time (1980s), Claimant was apparently "having shortness of breath and had pulmonary function studies done. The first time was in – the first reports that I have were in 1994 and, at that time, he did have a significant abnormality in his lung function testing." Dr. Baker testified that as of the 1994 test:

> [Claimant] had a restrictive and an obstructive abnormality at that point, though his obstruction was – at least my evaluation of his pulmonary function studies indicates that his obstruction was predominantly in the smaller airways because his –the FEV_1, FEC ratio there was normal which is sort of a *sine qua non* for obstruction. ... And he had an enlarged – or residual volume which suggests that he had some obstruction which means that his lungs – he couldn't exhale completely, the air flow was obstructed to get out of his lungs, and his total lung capacity was mildly reduced at that point, his diffusion capacity was normal.

(CX 6-6). After some confusion as to which results counsel was looking at, Dr. Baker was examining the May 13, 1994 test. (CX 6-7). At any rate, both of the tests show restrictive and obstructive elements. (CX 6-7). Dr. Baker added:

> [Claimant] probably had some small airways obstruction, and I made that on the basis of his elevated residual volume, which we were just talking about, means that he couldn't exhale all of the air out of his lungs. Residual volume being the amount of air that's left in the lungs after you've maximally exhaled so – but that not uncommonly occurs in people who have asthma, you see that they have an elevated residual volume.

(CX 6-8).

Dr. Baker also commented on lung volumes and restrictive impairment versus restrictive lung disease. He testified:

> Classically one sees with a restrictive abnormality a reduction in all lung volumes; however, if there's a superimposed obstruction involved in it, one can see that you don't have – that we were just talking about, the residual volume, can be increased, so what you have to look at is the entire picture since nobody fits the classic picture. (CX 6-8).

Dr. Baker noted that the fact that in both the 1994 and 1998 study the FEV_1, FEC ratio is normal and that, that is perplexing. He states "I've said [in my notes] several times I do not know why he has as much dyspnea as he does, because generally one would not see the level of problem that he has with the lung function test he has." (CX 6-9). In discussing the unusual nature of Claimant's dyspnea, Dr. Baker also notes that he has had a thorough cardiac evaluation and pulmonary exercise stress studies. (CX 6-9). *See also* (CX 6-10)(discussing the type of tests done by Dr. Ridley, who ruled out diastolic dysfunction in Claimant's heart with a heart catheterization).

Dr. Baker was also asked if he thought that Claimant's cardiac problem caused or contributes to his shortness of breath. Dr. Baker deferred to Dr. Ridley's opinion on that issue. (CX 6-11).

Asbestosis

Dr. Baker was asked to explain his note that the presence of a normal diffusion capacity on Claimant's pulmonary function study suggest that he does not suffer from pulmonary parenchymal abnormality. (CX 6-11, 12). He replied:

> Well, you know, at least I'm dealing with a patient who has significant asbestos exposure and who is short of breath, and you try to put those two things together and find out if he does have asbestosis. And as I've indicated in one of my previous notes, I could not make that diagnosis because I don't think he has some of the findings that are necessary to make the diagnosis. One of those is his chest

> does not have any crackle when I listen to it, and although he does have a restrictive component in his pulmonary function testing, his chest x-ray is not diagnostic of asbestosis, which is a fairly characteristic chest x-ray picture. He does have some scarring in his lungs, but I didn't think it rose to level that one would make a diagnosis of asbestosis.

(CX 6-12, 13). In order to clarify, Dr. Baker testified, there were two options a high resolution CT scan or a lung biopsy. He prefers to use the noninvasive procedure first. (CX 6-13). When asked if he was talking, at this time in his treatment and diagnosis of Claimant, about a possible sub-clinical asbestosis, Dr. Baker replied that he would need more information to diagnose and that he "hesitate[d] to use the term 'sub-clinical' because the man is so symptomatic." (CX 6-13).

Dr. Baker was next asked about Claimant's normal diffusion capacity. In an office note Dr. Baker apparently wrote "The presence of a normal diffusion capacity suggests that the problem isn't pulmonary parenchymal." Counsel asked if, generally, one of the precursors to even showing the lung scarring indicative of asbestosis on an x-ray was an abnormal diffusion capacity. (CX 6-13, 14). He replied:

> Well, we talk about classical findings of asbestosis and one would expect to see, pulmonary function testing-wise, restrictive abnormality and reduction in diffusion capacities, but asbestos is an odd disease. I guess it's like many others but you see a number of people who have a profusion of small irregular densities in their chest x-ray which is, say, one slash one with a normal diffusion capacity, and so that's not typical. But I've seen enough of them to say that, you know, that a percentage of them you do see that way and the – his –and the other end of that instrument also valid, that is do you see people who have [abnormal diffusion capacity] and no x-ray abnormalities of asbestosis or pulmonary fibrosis[.] Yeah, you can see that and particularly it's a problem in people who have underlying emphysema.

(CX 6-14). Dr. Baker testified that he did ascribe to the American Thoracic Society criteria for diagnosing asbestosis. (CX 6-14, 15). He agreed that, in their official statement, the Society indicates that reduction in diffusion capacity is "in a perfect world" one of the first things you'll see as someone gets asbestosis." Also, he agreed that according the AMA Guides, African-Americans do have lung volumes that are slightly smaller than same-sized Caucasians and so adjustments may have to be made to standard numbers. (CX 6-33).

Dr. Baker stated that the presence of pleural plaques is "a marker of asbestos exposure; it may or may not be related to asbestosis." While Dr. Baker refused to call pleural plaques clinically insignificant, as they show asbestos exposure, which warns you that you have a patient with a higher potential of having those other conditions that occur with asbestos factors. However, pleural plaques rarely cause clinical disability. (CX 6-15, 6-16). In the instant case, he states that Claimant does not have pleural plaques so Dr. Baker relied on Claimant's history in establishing that he has a history of significant asbestos exposure. (CX 6-16). Dr. Baker also commented on his other patients that had given him the same descriptions of exposure. (CX 6-16). Dr. Baker stated that he could not make a diagnosis of asbestosis. (CX 6-17). When asked: "And essentially, at this time, you don't believe that the problem is pulmonary parenchymal, is

that correct, from your statement?" Dr. Baker replied: "Well, I can't make that diagnosis with the information we have." (CX 6-17).

Dr. Baker also considered the idea of a chest wall abnormality and that Claimant has a beginning pulmonary fibrosis that is not readily assessable by standard chest x-rays. (CX 6-17). Dr. Baker explained that Claimant does not have the standard radiological criteria, pulmonary testing, and physical examination of one with asbestosis. (CX 6-18). Instead he states:

> [Claimant] has symptomatically a problem that because of his very thorough cardiac evaluation symptomatically he has something that looks like he has lung disease. Now I can't define it any better than I have for you, but he's a man who – at least if what he tells me is true and I have no reason to believe that it isn't – he was a guy who has worked in the Shipyard for most of his adult life, he never, smoke, he's never been around anything other than the things he worked with in the Shipyard which could have caused him to have lung disease and now he's disabled. He can't work and he can't work because he gets short of breath if he does anything, and you put two and two together and try to come up with four. But with that the problem is trying to put a name on what he's got. ...
>
> And it's very difficult in his case otherwise we wouldn't be talking, you know, if it were a cut- and-dry case you-all would have accepted it before.

(CX 6-19).

Employment related

Dr. Baker testified, that he could not say with complete certainty that Claimant's condition was caused by his employment. He further stated: "I have the sense that it was made worse by his employment." With a question of counsel, Dr. Baker clarified, that when he said he had a sense that it was made worse it is still uncertain what is being made worse, merely that it's dyspnea which is a symptom. Dr. Baker stated:

> He does have some airways disease and he does have some restrictive disease, whether it's parenchymal or not is the question we're chewing on, you know, and at that current moment it's very hard to make that... It's a diagnosis of exclusion. You'd have to look at the possible causes of chest wall restriction, which at least there's no evidence that he has that, you see that in people who had polio or who have other muscular diseases. You can see it in people who have severe spinal arthritis, ...who've got skin problems like scleroderma, or you can see it in people who've got a pleural disease, that is they've got lungs that can't expand because they've got pleural fibrosis. None of these are obvious as one examines [Claimant].

(CX 6-21, 22).

Dr. Baker stated:

> I think that working in a dirty, dusty, pain-filled, chemical-laden environment for anyone with airway disease is going to aggravate it, so if his work requires that he do that which it has in the past – to my understanding which it has in the past —then I think that has aggravated his airway problems.

(CX 6-23). Dr. Baker agreed that going out in the heat aggravated Claimant's symptoms as well. Dr. Baker clarified:

> Well, going out in the heat aggravates his symptoms, but going into a potentially chemical-containing environment or dirty, dust irritating environment could not just change the symptoms but could also make the airway process worse.

(CX 6-23). The following exchange then occurred:

> Q: You say could. Again, is that something that you can state with medical certainty not even knowing what the underlying condition is?
>
> A: If we want to talk in generic terms, we have a patient who has airway disease which responds to bronchodilators which is a form of asthma, asthmatic bronchitis, or whatever you want to call it. If that person is required to be in an environment which has irritants that he breathes in, yes, I think you can make that statement with complete certainty that that will aggravate and make worse the process.
>
> Q: But, again, we're talking hypothetically and generally. We don't know, in [Claimant's] specific case..., exactly what his underlying condition is and, therefore, how it can be aggravated with medical certainty.
>
> A: I think [Claimant] does have some airway disease and he does respond to bronchodilators though it's not great. And, in addition to that, he has a restrictive problem, and I think we have at least the two sets of pulmonary function studies that – you see the one from '94 at which time he had a very large residual volume which is an indication of air trapping which is an airway disease, and then the one in '98 not showing that suggests that he does have a reversible airways obstructive abnormality. Indicates – not suggests–indicates that he does.

(CX 6-24, 6-25). Finally, Dr. Baker stated that, although he didn't hear anything that made him think Claimant had lung disease when he listened to his lungs, that did not mean that there was no lung disease. Also, during examinations, Claimant had no acute dyspnea at rest. (CX 6-28).

Claimant told Dr. Baker that the last time he worked was in November 1998. (CX 6-29).

Dr. Baker indicated that Claimant reached maximum medical improvement in November of 1998. (CX 6-30). Dr. Baker did note that there are probably some jobs Claimant could do, in a sedentary position in a clean environment, and that Employer had no such jobs available as of the time he filled out his office note and report dated April 15, 1999. (CX 6-31). He also stated that he did not think that Claimant could do heavy manual labor. (CX 6-33). Another important restriction for Claimant's employment is a clean air environment. *See also* CX 6-34, 6-37(discussing employment options for Claimant). Finally, Dr. Baker was asked to discuss Claimant's work restrictions and the forms he filled out. He clarified, I think Claimant could stand for longer than one to two hours (if he could sit at his discretion), it's the "or walk" that made me check the one to two hour box. (CX 6-37, 38).

Reversibility

Dr. Baker was asked to comment on the two pulmonary specialists [Dr. Ross and Dr. Shaw] who opined that "any aggravation of any underlying obstructive or restrictive and mild obstructive or restrictive disease would be temporary in nature. That is it would subside and be reversible, it would subside after you take them away from that exposure whether it be dust, fumes or whatnot." (CX 6-25, 36). Dr. Baker replied that sometimes it did, sometimes it didn't. Specifically commenting on Claimant he stated "I think that he does have significant reversibility." (CX 6-26). Reversibility indicates a temporary process, however, the underlying process does influence this process. (CX 6-27).

Report of Claimant's high resolution CT of the chest, from Sentara Virginia Beach General Hospital dated September 20, 1999 and requested by Dr. Baker. The diagnosis and history both read "pulm fibrosis." The diagnosis reads:

> Nonspecific subpleural parenchymal scarring is demonstrated in both lung apices associated with focal areas of pleural thickening most likely post inflammatory in nature. There is also mild interstitial fibrosis and parenchymal scarring in the anterior segment of the right upper lobe associated with ectatic thickened bronchi representing cylindrical bronchiectasis. Mild cylindrical bronchiectasis also noted involving the posterior segments of the right and left lower lobes without significant associated parenchymal or pleural abnormality. No pulmonary infiltrates or mass identified and there is no evidence of pleural mass or effusion. Mediastinal images are not suitable for accurate evaluation, however, ectatic calcified aortic arch is demonstrated.

The impressions were:

> Mild post inflammatory parenchymal and pleural scarring in both lung apices.
>
> Mild interstitial fibrosis and parenchymal scarring in the anterior segment of the right upper lobe associated with segmental cylindrical bronchiectasis.
>
> Mild cylindrical bronchiectasis involving the posterior basal segment of the right lower lobe and left lower lobe and to a lesser extent lingula without significant

parenchymal abnormality.

(CX 2J-A).

In a letter to Dr. Acosta dated September 22, 1999 from Dr. Baker, he reports on Claimant's high resolution CT scan. Dr. Baker writes:

> I am somewhat surprised to find that [Claimant] does have significant upper lobe cylindrical bronchiectasis. That usually is in response to a significant pulmonary infection and I don't remember [Claimant] telling me that he had had such. I am again perplexed by [Claimant's] information, however, this is certainly something which could partially explain his symptoms although I am not sure why it explains his restrictive lung abnormality.

(CX 2I-A).

In a letter to Claimant's counsel dated October 5, 1999, Dr. Baker comments on possible jobs for Claimant. He writes that generally he believed a parking lot attendant at the Norfolk Airport Authority, cashier at a Crown gas station, counter person at Firestone would be acceptable. He did state that the job at Nor-Ship Co "would have to be seen personally to make a decision...." (CX 2K-A), (EX 27).

In a letter to Dr. Acosta dated November 10, 1999, Dr. Baker updates him on Claimant's condition. (CX 2L-A). Again, Dr. Baker uses the shorthand of S.O. A. P., which has not been explained to the court. It is clearly noted, however, that Claimant returned to Dr. Baker's office with the "chief complaint of continued shortness of breath." During his examination Dr. Baker noted, for the first time, hearing bibasilar inspiratory crackles which do not clear with deep breathing and coughing." (CX 2M-A). Under A., Dr. Baker writes:

> 1. Rhinitis and postnasal drainage
> 2. Restrictive lung disease etiology undetermined
> 3. Mild obstructive airways disease etiology undetermined both probably related to his occupation.

(CX 2M-A). Dr. Baker ends by stating: "I am still concerned regarding the exact etiology of [Claimant's] pulmonary problem but again I believe that in a nonsmoker who worked for approximately 30 years in the shipyard that his problem must be occupationally related." (CX 2M-A)

The radiology report regarding the study of Claimant's chest, dated March 20, 2000, and signed by Nripendra Devanath, M.D., indicated that mild pulmonary emphysema was possible due to mildly hyperinflated lungs. The study showed that the lungs were free of any acute or active process. Apparent mild cardiomegaly was also noted. (CX 2-6). *See also* (CX 2-7, 2-8), (EX 26).

The office notes of Dr. Eric Freeman indicate that Claimant visited him on March 20,

2000 complaining of chronic shortness of breath. He notes that Claimant had an extensive evaluation from Dr. Baker. (CX 2-3). After a physical exam, review of Claimant's medical and social history, and review of several tests, Dr. Freeman stated that Claimant had a restrictive lung defect documented on several pulmonary function tests, the cause of which was unclear. Claimant was found to have bronchiectasis, however he is totally asymptomatic. He stated that "[t]he amount of pulmonary fibrosis on his CT scan I doubt could account for [his restrictive lung defect] although for now I think we must attribute his restrictive lung disease to a localized fibrosis." (CX 2-4),(EX 29).

In Dr. Freeman's office notes, dated April 5, 2000, Dr. Freeman stated that Claimant continued to be short of breath and tightness in his chest when he walks at times. (CX 2-2.). After a physical exam, Dr. Freeman was still puzzled about the problem. He noted that there was "equal and symmetrical expansion of the chest; no dullness to percussion with an absence of wheezes, rales or rubs." Dr. Freeman expresses puzzlement as to the cause of Claimant's complaints. He writes, "I doubt if he has pulmonary hypertension with complaints going back to 1994 and a recent cardiac catheterization and echocardiogram." (CX 2-2), (EX 28-1). He found no significant abnormalities in Claimant's physical exam, CT scan or PFT to suggest the symptoms he reports. His plan is to see Claimant again after reviewing all of his test results from Dr. Ridley. He writes "If pulmonary hypertension has not been ruled out, we will obtain a repeat echocardiogram to rule out pulmonary hypertension. If this is negative, we will proceed with advancing towards pulmonary rehab and psychological treatment." (CX 2-2), (EX 28-1).

Dr. Ross sent a letter to Employer's counsel dated April 20, 2000. After reviewing Claimant's medical records, provided by counsel, and preparing the requested reports, he opined:

> 1. It is my firm opinion that [Claimant] has a longstanding history of chronic bronchial asthma which was present before he began work [with Employer] which is, in itself, a permanent condition.
>
> 2. I agree that his overall impairment is due in principal part, if not exclusively, to his bronchial asthma.
>
> 3. I agree that, if he did not have the pre-existing bronchial asthma, he would have very minimal impairment, if any, attributable to occupational exposure.

(EX 21).

Dr. Eric Freeman states, in his office notes of May 26, 2000, that Claimant was in his office for an evaluation due to shortness of breath. (CX 2-1), (EX 28-2). Dr. Freeman stated that Claimant's past lab data indicate that Claimant had a restrictive lung defect, but no diffusion capacity abnormality and no airflow abnormality. *Id*. Claimant has a right coronary artery defect, but no pulmonary hypertension, no sign of congestive heart failure or any significant ischemia. Claimant also had an exercise study showing a defect, but the cause was unclear, perhaps cardiac, but that was ruled out by cardiac catheterization. *Id*. Dr. Freeman indicates that what is left is the fact that Claimant has a very mild lung restrictive defect of unknown etiology and a lot of symptoms of shortness of breath. He stated that there was very little that could be done unless

Claimant's pulmonary function worsened and he developed some type of restrictive defect which can be identified. *Id*. He noted that, even on a high resolution CT of the chest there was no evidence of pulmonary fibrosis. Dr. Freeman, therefore, started Claimant on pulmonary rehabilitation and restricted his activity because of the restrictive lung disease of unknown origin. *Id*.

Dr. Eric Freeman gave a deposition dated June 7, 2000, entered into evidence at CX 10. Dr. Freeman is a specialist in pulmonary medicine, Board Certified in Internal Medicine, and is a NIOSH B reader. (CX 10-3, 4). *See also* (CX 10- 51)(Deposition Exhibit #1, Dr. Freeman's Curriculum Vitae). Claimant was referred to Dr. Freeman after Dr. Baker retired. (CX 10-5). The first time he saw Claimant was March 20, 2000. (CX 10-6). At that time he had a chest x-ray for comparison done on Claimant, along with reviewing all of his previous extensive lab tests. (CX 10-6). Dr. Freeman was asked, based on all of Claimant's records, his own examination of Claimant, and any tests that he had performed, what conclusions he reached regarding the nature of Claimant's pulmonary condition. He replied:

> He has a restrictive lung disease characterized by a persistent restrictive lung defect on his pulmonary function test for – since the early 1980s which has been consistently abnormal. And I cannot characterize it any further than that. He hasn't submitted to a lung biopsy and I wouldn't recommend one, actually because it's – but there is no way to absolutely characterize the cause of his restrictive lung disease.

(CX 10-7). Dr. Freeman reviewed what Claimant had told him about his employment history in the 1970s, exposure to dust, asbestos, and fumes. He then opined, to a reasonable degree of medical certainty that Claimant did not have asbestosis. (CX 10-8).

Dr. Freeman stated that he did review Claimant's pulmonary function tests from as far back as the 1980s and that those tests also showed "this restrictive lung defect which has been pretty consistent in all of his pulmonary function tests." (CX 10-8). In fact, specifically the test of July 10, 1981 showed a restrictive lung defect. (CX 10-9).

Dr. Freeman was asked about possible causes of Claimant's "restrictive lung defect." He replied:

> There are many causes for restrictive lung defects including inhaling dusts and –but in this case, I – I feel it's not asbestos dust because it would be – the latency period would be too short. Nineteen eighty-one from when he started working certainly wouldn't be twenty years, and that would be what we normally expect before we start seeing restrictive lung defect from asbestosis, unless he had extensive pleural
> effusions which can occur earlier. But he has a fluoroscopy which shows no evidence of pleural effusion. So there would be no reason for him to have as – we could almost positively rule out asbestosis as the cause. What other dusts he's inhaled which could cause this, I'm not sure. I mean, there are many dusts that you can inhale that can cause asbestosis, plus there – I mean, restrictive lung

> disease, plus there are other things such as .... [a heart condition]..only if he was in congestive heart failure at the time, that could mimic that. But he couldn't be in congestive heart failure for twenty-some years. ...

(CX 10-9, 10). Dr. Freeman testified that Claimant reported to him that his breathing problems worsened in the nineties, specifically that he says now he feels incapacitated at times. (CX 10-11).

Dr. Freeman was also asked to comment on Claimant's high resolution CT. Although Dr. Freeman could not locate the report he did find Dr. Baker's impressions of that CT scan, which indicated significant upper lobe cylindrical bronchiectasis, indicative of a significant infection. Dr. Freeman stated:

> And I don't remember [Claimant] telling me he had that. Matter of fact, I also saw this. I'm not sure it was cylindrical bronchiectasis but he did have some – some changes in his upper lung field that could be cylindrical bronchiectasis. But the fact is that is accompanied by symptoms of large amounts of sputum production on a chronic basis. He had none of those symptoms. He had no fever, no bleeding or anything to suggest bronchiectasis.

(CX 10-12, 13). Dr. Freeman also stated that the scan showed no findings consistent with asbestosis. (CX 10-13).

Dr. Freeman testified that he had, in his practice, encountered other individuals who worked as marine welders, who had not been exposed to asbestos and were not smokers, who developed breathing and lung impairments. (CX 10-14).

In the case of Claimant, assuming he does not suffer from asbestosis, which Dr. Freeman feels quite certain that he does not, considering his entire medical and employment history, the most likely cause of his restrictive lung defect disease is "some form of parenchymal lung disease that we're unable to characterize." (CX 10-15, CX 10-16). Dr. Freeman testified that he could not say what would be the causative agent, although he did state that "the interstitial lung disease, the lung itself being involved is the most likely cause of his restrictive lung defect.... (CX 10-16).

Dr. Freeman was asked to opine, based on all of the information he has, as to what role his exposure to dusts, smoke, fumes, and other matters who would have breathed as a welder over the years played in the development of his problem. He replied:

> This is the most likely source of his lung disease. There is no other obvious source. He does not have other diseases that we can tell that cause restrictive lung disease such as sarcoidosis. He does not have any evidence of that. He has no evidence of lupus or any other immunologically related lung disease that causes a restrictive lung defect. So the assumption, then, would be that it would be from inhalation of certain unknown particles that are causing his interstitial lung disease.

(CX 10-17). Dr. Freeman states that he is able to focus on this exposure rather than the asbestos exposure during the seventies because:

> Because if he had – if this is due to asbestosis, then his restrictive lung defect would not show up for another ten years but it was already present in 1981, document on this–in this workup at Duke University. Asbestos dust inhalation has a time to when you develop the parenchymal lung disease. The latency period is twenty years. And he didn't have a latency period of twenty years, not even close. ... [C]ertain inhaled substances can cause a restrictive lung disease much sooner... for instance silica.

(CX 10-17, 18).

Dr. Freeman was also asked about the painting that Claimant reported during 1996 and 1998, however, he could not pinpoint it as a source unless he knew what was being ground up, although it sounds logical. (CX 10-18,19).

Dr. Freeman was asked if, in his opinion, there was any discrepancy between the severity of the symptoms Claimant reports and his defect, which is characterized as mild to moderate. (CX 10-19). He replied that shortness of breath is a subjective, and therefore variable, symptom and people have different tolerances, emotional responses, and physiological conditions. (CX 10-20).

The following exchange occurred:

> Q: If we understood the term reasonable medical certainty to mean that it was just more likely than not, if we had a particular proposition and we were to determine that it was a greater than 50 percent likelihood that that was the case, it was true or accurate, in his particular case you've indicated that the various possible causes, musculoskeletal, heart, asbestos, and his longterm exposure to dusts, smoke, fumes, and other inhalant matters at the shipyard, that that would be the most likely cause?
>
> A: Yes.
>
> Q: Given that definition of more likely than not, do you feel that–that in your opinion you can state that in his case?
>
> A: Yes. And, basically, because we can't find any other cause for his restrictive lung disease, that would–that would be a logical explanation.

(CX 10-20, 21).

Dr. Freeman was then asked about the dusts and exposures Claimant might find outside of

work, and he stated that, if his hobbies were unusual, such as sandblasting, mining, grinding, on his own, there would be this type of exposure. (CX 10-22). Essentially, there are very few things that mimic or cause restrictive lung disease; it would be "unusual to find a hobby to cause this kind of condition. It's possible, I guess, but it would be unusual." (CX 10-23). Employer takes issue with the "speculation" that Claimant inhaled substances that may cause this lung defect at work. (CX 10-23, 24).

The following exchange then occurred:

Q: Presently he has a restrictive ...lung disease...that would mimic asbestosis but for a latency period?

A: Exactly. ... But there are others [sic] things that make us feel that he doesn't have asbestosis as well ... his x-ray, his CAT scan, and so forth. (CX 10-24)

Q: The pulmonary function studies, though, are consistent with asbestosis too?

A: Absolutely.

(CX 10-24, 25). Dr. Freeman agreed that there are some findings that are consistent with asbestosis, just not a whole range of them that all fit together, but he feels that there is enough to rule it out conclusively. (CX 10-25). Dr. Freeman was then asked:

Q: But if you're standing here thirty years–if you didn't have the pulmonary function study in 1981 that said restrictive lung disease –I'm sorry –restrictive impairment ...you would be sitting here with a thirty year latency period ... a history of exposure to asbestos dust, a restrictive lung impairment, and pulmonary function studies that bear it out, would you probably make the diagnosis with that?

A: I couldn't but I would not be able to exclude it. ... And the reason I couldn't is because he doesn't have the characteristic x-ray findings. But I –but there are patients who can have pulmonary fibrosis which are not picked up on chest x-rays. We do –we do sometime detect restrictive lung disease and they do that asbestosis when we do a biopsy, for instance. And –but, clearly, because we have this pulmonary function test, I don't think that there's any question even –as atypical a case it would be, even thirty years without the test, now with that it would be incomprehensible that he had asbestosis.

(CX 10-25, 26). Dr. Freeman explains that if Claimant had a restrictive lung disease and exposure to asbestos dust, it could have a very profound effect on his condition or none, depending on if he developed asbestosis. (CX 10-26). Inhaling asbestos dust by itself does not cause asbestosis, so if he inhales the dust and already has restrictive lung disease, it certainly would not make his

restrictive lung disease any better, it might make it worse, or it might not make it any worse. (CX 10-26, 27). It would only combine to cause a greater restrictive impairment if he developed asbestosis. (CX 10-27).

Dr. Freeman was then asked, if two dusts that cause restrictive impairment combine over a period of time, if the effects or cause of a restrictive impairment can really be separated out. He replied that, with a reasonably degree of certainty you could in this case because Claimant already suffered a defect in 1981. That defect has not changed much and he has no evidence of asbestosis by any of the usual way of diagnosing asbestosis-except for that one pulmonary function test which was abnormal and very little unchanged. (CX 10-27, 28).

Counsel then laid out the following facts: Claimant's 1981 pulmonary function study showing restrictive impairment; exposure to asbestos dust in 1970s on a regular basis; and in 1998 he has a restrictive impairment. He asks:

> Q: Now you're targeting as the cause of his incredibly increased symptoms in the 1990s, that was a pretty dramatic change from ... being able to... [his symptoms, not his pulmonary function test changed, his test did not change that much]
>
> A: ...symptoms don't always correlate with the actual lung dysfunction that we detect on pulmonary function testing.
>
> Q: And asbestosis can exists without having any definitive clinical evidence of it, right?
>
> A: Well, ...it's possible [with no clinical evidence, no pulmonary function studies showing restrictive lung impairment]. ... But it can't be diagnosed without at least some abnormality, either characteristic x-ray findings and a history of asbestos dust exposure or, in –in some cases, significant restrictive lung defect and –which develops at a proper period of time after exposure to asbestos dust. But then you might –you couldn't actually say the patient definitely has asbestosis but you could say that the pulmonary function test possibly is causing the asbestosis [sic?]. But this is not the case with this gentleman. In this case he developed the – or at least he had documented restrictive lung defect long before he could have developed asbestosis. The lung defect itself, as judged by his pulmonary function test, has remained relatively stable. And that to me would – clearly indicated that asbestos dust inhalation and the development of asbestosis is not an issue here.

(CX 10-30 thru 10-32). He agreed that we are not able to identify the other dusts, and their latency period, that Claimant may have been exposed to. (CX 10-32).

Dr. Freeman reiterated that he could, with a reasonable degree of medical certainty say

that Claimant suffers from an occupational disease. He clarifies:

> All I can say is the most likely cause for this condition, and that's all I ever said, was that it was probably what he–something that he inhaled while employed, wherever he was employed, at the shipyard. And because of the fact that this occurred without any other obvious causes
> --
> causes –restrictive lung defects and restrictive lung diseases related to restrictive lung defects are relatively uncommon. Most of the diseases we see that are chronic diseases are what we called obstructive lung diseases. And so restrictive lung diseases are very uncommon. And their causes are usually fairly obvious from either rheumatoid arthritis associated diseases, or connective tissue diseases, a disease called sarcoidosis, certain drugs that people take that can cause these kinds of condition[s] like gold injections and stuff like that. So to see something like this is unusual. And if there's no other explanation for it, then this would –then an inhalation cause would be the most likely explanation for this. And that's all I can say. I can't say with a 100 percent certainty but it would certainly be the most likely explanation.

(CX 10-32,33).

Dr. Freeman had not, until this date, seen any tests or impressions stating that Claimant suffers a "mild obstructive" impairment. He states that he would disagree with that interpretation, with great confidence, due to Claimant's good flow pattern ability. (CX 10-34). Dr. Freeman also commented on Dr. Zimmet's opinion, a mixed defect but based on different radiological evidence. The x-ray used here was evaluated by Dr. Harron, a B reader, whose interpretation leaves room for debate. Again, there are no pleural plaques or pleural involvement. (CX 10-36, 37). He also doubts that Dr. Zimmer saw Claimant's 1981 test, again focusing on the latency period of asbestosis. (CX 10-39). See discussion of latency period CX 10-39 thru 41).

When discussing latency periods, Dr. Freeman elaborates:

> And, like I said, I think that there are always biological variants between people but we're talking [in Claimant's case of cutting the latency period in] about half. And that would be extremely unusual. And then with the absence of pleural plaquing and the absence of characteristic findings on the –on the CT scan, I think that asbestosis is not a consideration.

(CX 10-41). Dr. Freeman did admit that it was possible Claimant had silicosis or siderosis although "we don't see [either of them] on the x-rays." (CX 10-41). He states: "sometimes you can get interstitial lung diseases and it doesn't show up on x-ray, it just shows up on pulmonary function tests." (CX 10-41, 42).

Dr. Freeman agreed that usual interstitial pulmonary fibrosis is an idiopathic condition, meaning it has no known cause. (CX 10-49). He stated, however, that it "would be extremely unlikely [in this case] because the course of the condition usually is progressive and fatal within

five to seven years from the onset, also the lack of findings on CAT scan to support that diagnosis." (CX 10-49). He does acknowledge that there are cases where it could last for much longer periods of time. (CX 10-49).

Dr. James Baker testified in a post-hearing deposition taken on October 8, 2002, (CX 13) that he does not believe that the Claimant has asbestosis, that any impairment that he does suffer is not related to asbestos exposure, but that such impairment is related, instead, to occupational exposures to dust or fumes. However, he also testified that Mr. Richardson has asbestos exposure and has some symptoms [of asbestos disease], and that he has lung function tests which are compatible with asbestosis. But, "without x-ray evidence of pulmonary fibrosis, I don't think you can make a diagnosis of asbestosis."

On cross examination, Dr. Baker acknowledged that the Claimant does have a reliable history of asbestos exposure, and that an appropriate time interval between exposure and detection has occurred. Further, Dr. Baker states that the Claimant does have a restrictive pattern on his lung function test, consistent with asbestosis. He also testified that, while he read the Claimant's x-ray as negative for asbestosis (0/1), Dr. Zimmet and Dr. Harron both read Claimant's November 13, 1997 x-ray as showing bilateral pulmonary fibrosis, consistent with asbestosis. Dr. Baker agreed that there can be significant inter-observer variation in many x-ray reading tests, this one in particular, even variation by the same reader at different times.

Dr. Baker also repeated his testimony in his earlier deposition, that he really can't put a name to the Claimant's impairment, that he has exposure in his occupation, and he has not had exposure in other places, and that he is now impaired. "And, again, I would say that I think that one and one equals two under those circumstances." When asked if his opinion as to the cause of Claimant's impairment was the same in 1999 as it is today, he states that,

> My opinion is, which I've stated already today, is that Mr. Richardson was a gentleman who worked in an environment that could produce inhalant lung injury, and from a number of various mechanisms. And he worked for thirty years, and then something happened to him and he no longer is able to work because he gets short of breath when he does anything.
>
> He does have abnormalities of lung function, and he does have some structural abnormalities in his lungs. He is a nonsmoker, and he doesn't have a significant, to my mind, history of lung disease otherwise. Therefore, I think that his problem is related to his employment.

Finally, Dr. Baker testified that he has seen people who have had asbestos exposure who have pleural plaques, markers of asbestosis exposure, who have abnormal lung function who do not have the chest x-ray abnormalities.

Dr. Eric Freeman was also deposed post hearing on October 8, 2002. (CX 14). He testified that the Claimant's CAT scan show focal fibrosis, but that it was a very small amount and did not account for the Claimant's shortness of breath. He testified that "I really did not address specifically that he had pulmonary asbestosis, but I did not feel at that time, I did not make a

diagnosis of pulmonary asbestosis." He also states that he did not diagnose pleural plaques by x-ray or CT. Dr. Freeman did acknowledge that the Claimant had a restrictive lung defect shown by pulmonary function test, that could possibly be ascribed to previous asbestos exposure. However, he states that

> "[I] could not account for that, certainly not from the degree of fibrosis on his CAT Scan. In order to have a restrictive lung defect for pulmonary fibrosis, you generally expect a reasonable amount of pulmonary fibrosis. But the fact is that he did have a restrictive lung defect on his pulmonary function test, and I believe I put in there that I was puzzled by the presence of that and the concern it had.

(CX 14, pp. 8-9). Nevertheless, Dr. Freeman testified that he did not believe that asbestos is responsible for the Claimant's restrictive lung disease.

The following exchange then occurred:

> Q. Doctor, just so the record is perfectly clear, is it your opinion that Mr. Richardson does not have pulmonary asbestosis?
>
> A. You know, from the chest X-ray, from the CT Scan, I can find no supporting evidence for pulmonary asbestosis. Or, let me put it this way: I cannot find with a resonable degree of medical certainty that he has pulmonary asbestosis. I think there are some things that – he has a history of asbestos dust exposure; he has this CAT Scan which shows tiny bits of fibrosis, but I think with a reasonable degree of medical certainty I think he does not have the asbestosis. This is not the purest picture.

(CX 14, p. 10). However, as to what would cause the Claimant's breathing impairment, if it is not asbestos, Dr. Freeman states that he did not know.

DISCUSSION OF LAW AND FACTS

Asbestos claim

Upon commencement of the hearing, Counsel for the Claimant in his asbestos claim, Mr. McCormick, stated that he wished to amend his asbestos related disease claim as follows:

> Your Honor, our position is that Mr. Richardson was employed at Newport News Shipbuilding and Dry Dock Company for a period of time, beginning in the '60s and ending in the '90s. And during that time frame, he was exposed to asbestos while performing his duties. By virtue of that exposure and evidence indicating that he's developed pleural plaques, pleural thickening as the records suggest, it is our position that he is entitled to medical monitoring under Section 7 of the Act.

> [And that's the] sum total of our claim in that matter. We do not believe that Mr. Richardson, right now, has Asbestosis, although he might in the future. He doesn't at this time.

Tr. 6-7.

Therefore, I find that the Claimant, at the formal hearing on the record, has withdrawn his request for compensation due to asbestos related disease, and has amended his claim to seek only an award for medical monitoring under § 7 of the Act due to asbestos related disease. (Tr. 5-7).

Additionally, during the hearing, Counsel for Employer and the Claimant (McCormick) stipulated that as a result of Claimant's exposure to asbestos, he is at heightened risk to have asbestosis, mesothelioma and lung cancer in the future. (Tr. 40-41). On cross examination, Dr. James Baker responded to Mr. McCormick that the Claimant, due to his exposure to asbestos, should be followed on a periodic basis by virtue of his previous asbestos exposure. (Tr. 72).

The Claimant has offered at least some medical evidence which contains the diagnosis that he has developed pleural thickening. See, (CX 2J-A), (CX 3C-A), (EX 9-4). However, prior to addressing the matter of entitlement to medical expenses, the issue of whether § 33(g) of the Act bars an award of medical expenses (medical monitoring) must be addressed.

Section 33(g)

Employer argues that Claimant's claim for asbestos medical monitoring is barred under the Act by 33 U.S.C. § 933(g)[7], because Claimant received third-party settlements with asbestos manufacturers without Employer's prior approval. (Employer's brief at 27.)

Section 33 is generally designed to foreclose injured employees from double recoveries where they receive both benefits under the Act and civil damages from a successful negligence action. Section 33(g) provides a bar to claimant's receipt of compensation where the person entitled to compensation enters into a third-party settlement for an amount less than his compensation entitlement without obtaining employer's prior written consent. 33 U.S.C.

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[7] Section 33(g) provides in relevant part:

> (1) If the person entitled to compensation (or the person's representative) enters into a settlement with a third person referred to in subsection (a) of this section for an amount less than the compensation to which the person (or the person's representative) would be entitled under this chapter, the employer shall be liable for compensation as determined under subsection (f) of this section only if written approval of the settlement is obtained from the employer and the employer's carrier, before the settlement is executed, and by the person entitled to compensation (or the person's representative). The approval shall be made on a form provided by the Secretary and shall be filed in the office of the deputy commissioner within thirty days after the settlement is entered into.
> (2) If no written approval of the settlement is obtained and filed as required in paragraph (1), or if the employee fails to notify the employer of any settlement obtained from or judgment rendered against a third person, all rights to compensation and medical benefits under this chapter shall be terminated, regardless of whether the employer or the employer's insurer has made payments or acknowledged entitlement to benefits under this chapter.

§ 933(g); *Estate of Cowart v. Nicklos Drilling Co.*, 505 U.S. 469 (1992). The section is intended to ensure that employer's rights are protected in a third-party settlement and to prevent a claimant from unilaterally bargaining away funds to which employer or its carrier might be entitled under 33 U.S.C. § 933(b)-(f). *See I.T.O Corp. of Baltimore v. Sellman*, 954 F.2d 239 (4th Cir.), *modified on reh'g*, 967 F.2d 971, (4th Cir. 1992), *cert. denied*, 507 U.S. 984 (1993). Employer bears the burden of proving that claimant entered into fully executed settlements without its prior written approval in order to bar claimant's receipt of future benefits as Section 33(g) is an affirmative defense. *Barnes v. Liberty Mutual Ins. Co.*, 30 BRBS 193 (1996).

The record contains evidence that the Claimant was involved in litigation against asbestos manufacturers due to his exposure to asbestos, and that he entered into settlements in two cases. Employer offered as EX 6, correspondence addressed to the Claimant and his wife from Peter T. Nicholl, Claimant's Attorney in two asbestos litigation cases. The first letter, dated February 9, 1999, references an offer of settlement from "Babcock & Wilcox" in the amount of $6,500.00, and recommended that Claimant accept the settlement amount. (EX 6 p. 3). The second letter, dated June 17, 1999, indicates that a check in the amount of $74.67[8] was enclosed for "Forty-Eight Insulations Settlement." Mr. Nicholl states that this check represents payment of a settlement of "$112 less our 1/3 attorney's fee." (EX 6).

Claimant's Counsel (Walsh) has acknowledged that the Claimant entered into a compromise settlement of his asbestos claim against a third party manufacturer for $78.00 (sic), but that he never signed a release for the $78.00 check. (Claimant's brief at 27.) Claimant testified that he received a check for $78 and some cents from Attorney Nicholl in an asbestosis settlement, but that he did not sign a release. (Tr. 132-133). However, the June 17, 1999, letter from Attorney Nicholl advised the Claimant that "The amount is so nominal because Forty-Eight had very little in assets to divide among tens of thousands of claimants. Under the terms of the Trust Agreement, it was not even necessary to submit a release to be a part of this settlement." (EX 6, p. 1).

Upon consideration of this evidence, I find that the Claimant's acceptance of the check in combination with the letter from Attorney Nicholl, is sufficient to establish that the settlement for $112.00 ($74.67 net to Claimant) with "Forty-Eight Insulations" was fully executed.

Employer also argues that Claimant entered into a fully executed settlement with Babcock and Wilcox for $6,500 in settlement of litigation. (Employer's brief at 29.) In support of this assertion, Employer submitted a copy of a "Release and Settlement of Claim" dated February 16, 1999. This document is signed by the Claimant and his wife, acknowledging receipt of $6,500.00, and releasing "The Babcock and Wilcox Company" from all liability relating to Claimant's asbestos exposure. (EX 24).

However, Claimant's Counsel (Walsh) argued in his brief that "Second, claimant never consummated any other settlement other than the seventy-eight dollar asbestosis claim, thus Section 933(g) is not applicable." (Claimant's brief at 28.) The Claimant acknowledged that he

[8] The Claimant and his attorney erroneously refer to this check as being in the amount of $78.00 dollars.

signed the settlement and release, and that the money came to his attorney (Nicholl). However, after having signed the release and acknowledgment, he says that he was advised by his worker's compensation attorney that it was not in his best interest to accept the money. Therefore, Claimant testified that he directed his attorney to cease representing him in the asbestos litigation. (Tr. 134-137).[9]

Upon consideration of this evidence, I find that the Claimant fully executed the settlement with Babcock and Wilcox for $6500.00. Although the Claimant subsequently refused the check, such does not negate his execution of the settlement agreement which authorized his attorney to receive the funds, and the attorney did, in fact, receive the funds.

Further, it is undisputed that the Employer did not give its written consent to settlement of either case (Tr. 134). Thus, I find that the Employer has met its burden of proving that the Claimant entered into fully executed settlements of claims against asbestos manufacturers (on February 16, 1999 and June 17, 1999) without its prior written approval.

In order for the § 33(g)(1) bar to apply, Claimant must be a person entitled to compensation [at the time he entered into a 3rd party settlement]. The Supreme Court has also addressed the issue of who is a "person entitled to compensation" within the meaning of § 33(g) in its decision in *Cowart, supra*. The Court held that a person entitled to compensation need not be receiving compensation or have had an adjudication in his favor [to be considered a person entitled to compensation]. Instead, the Court held that a person needs only to have satisfied the prerequisites attached to the right. Further, the relevant time for examining whether a person is "entitled to compensation" is the time of settlement. *Id*. Therefore, the evidence in existence at the time of the settlement must be considered in determining whether the Claimant was a person entitled to compensation.

Under § 20 the Act, if the Claimant shows that he suffered a physical harm and an accident at work or conditions at work, which could have caused the harm or pain, it is presumed that the accident caused the harm. It is undisputed that the Claimant was exposed to asbestos as a welder for many years (1968-1980) while working for the Employer, and that this employment was "covered" under the Longshore Act. As of February 9, 1999, the date of the "Babcock & Wilcox" settlement, there was medical evidence in existence that the Claimant had asbestosis and pleural thickening.[10]

A medical note from Dr. Herbert A. Saltzman of Duke University, dated July 13, 1981, indicates that the Claimant had exertional dyspnea since 1979 and that such might be related

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[9] Apparently, the funds remain in escrow in the matter pending before the Circuit Court for the City of Portsmouth, Va. (EX 25).

[10] It is noted that evidence subsequently developed, when weighed with the evidence in existence at the time of the asbestos negligence settlements, might or might not result in a finding that the Claimant does not have asbestosis. However, that is not the issue when considering the application of § 33(g), as only the evidence in existence at the time of the settlement is relevant. The reason for this is that the Employer's right to defend its claim of subrogation in the negligence action has been terminated by the Claimant's settlement without its knowledge and consent.

either to significant occupational exposure to asbestos or pre-existing asthma. (CX 12-11,12.)

In a report dated January 31, 1994, Dr. Stephen A. Fink (or Pink) interpreted Claimant's chest x-ray. His impression was that there was no acute process and he noted that there were no previous films for comparison. He also noted that "[c]hest examination shows the heart size is normal and the lungs are well aerated and clear. Minimal bi-apical pleural thickening is noted." (EX 9-4).

On a medical summary form of Employer it is noted that on June 24, 1994, Claimant was diagnosed with asbestos related lung disease. There is no signature. (EX 10-47).

In a letter to Dr. Acosta from Dr. Childs, dated June 26, 1994, Dr. Childs states that Claimant was seen initially on June 17, 1994 after six months of complaining of shortness of breath associated with increased dyspnea while climbing stairs and some chest tightness with exertion. (CX 1-7). Among other factors, Dr. Childs noted that Claimant "actually had a severe obstructive defect and a mild restrictive defect with the mild reduction in his TLC," and [t]here was minimal biapical pleural thickening." (CX 3C-A). Dr. Childs' initial impression was that Claimant had severe chronic obstructive pulmonary impairment, but he could not tell if it was a significant component because no bronchodilator was given. (CX 1-8). He noted that he was concerned about Claimant's abnormal cardiology work-up and stated "[t]hese [cardiac] changes would make me concerned that the patient's obstructive defect might be fixed." Dr. Childs adjusted Claimant's medications, advised him that he would need another full PFT work-up and referred him to a cardiologist. (EX 1-8). *See also* (CX3C-A),(CX3D-A), (EX 12-6, 12-7).

In a report of occupational illness form dated March 5, 1995, Claimant states that he was injured by "inhalation of airborne asbestos dust and fibers." It is noted on this form that the reporting date was February 20, 1995, the injury date is noted to be June 24, 1994, and it is noted that it is controverted as not arising out of or in the course of employment. The illness is noted as asbestos related lung disease and the name of the doctor is Dr. George C. Childs, Jr. (EX 10-54).

In a report dated January 20, 1998, Dr. Ray A. Harron apparently reviewed Claimant's chest x-ray dated November 13, 1997. His impressions of those x-rays is that they are "consistent with asbestosis." (EX 23).

In a letter to Claimant's asbestos litigation counsel Mr. Nicholl, dated October 9, 1998, Dr. Steven M. Zimmet reported on his examination of Claimant on September 28, 1998. (EX 22). On that day Claimant had complete pulmonary function tests in addition to a history and physical examination. Dr. Zimmet also reviewed Claimant's chest x-rays. Under the heading "Data" Dr. Zimmet noted that Claimant's November 13, 1997 x-ray shows "increased pulmonary parenchymal markings indicative of bilateral interstitial pulmonary fibrosis. These films were also reviewed by Ray A. Harron, M.D. who agrees they are consistent with asbestosis." Dr. Zimmet also opined:

> Pulmonary function tests demonstrate a mild decrease in flow and vital capacity with a mild increase in residual volume and a moderate decrease in diffusing

capacity. These test results are indicative of a mild obstructive lung defect with a mild reduction in vital capacity and a moderate reduction in gas exchange.

(EX 22-2). Based upon Claimant's occupational history of asbestos exposure, abnormal pulmonary function tests and abnormal chest x-rays, Dr. Zimmet's assessment is that Claimant suffers from asbestosis. (EX 22-2).

Based upon this evidence and the Claimant's exposure to asbestos, I find that there was sufficient evidence, at the time of the third party settlements, to invoke the presumption of § 20 that Claimant's exposure to asbestos caused asbestosis and pleural thickening. However, to be entitled to compensation, the Claimant must then show that he has suffered a disability (loss of wage earning capacity) in order to satisfy the prerequisites for an award of compensation under the Act.

The parties have stipulated that the Claimant has been paid no workmen's compensation disability benefits as a result of his injury, but he has received Sick and Accident Benefits in the amount of $5,200.00 for the period covering 11/17/98 through 5/17/99, and the Employer is entitled to a credit for these monies against any compensation awarded for this period. Further, the Claimant is asserting a claim of temporary total disability for the same period, and a claim for permanent total disability thereafter. The parties have also stipulated that the claimant's average weekly wage at the time of the injury [November 18, 1998] was $820.20 resulting in a compensation rate of $546.81. Thus Claimant himself alleges 26 weeks of temporary total disability for a total compensation of $14,217.06, plus permanent total disability thereafter at the same rate. The Claimant credibly testified that he "came out" of his employment with Employer on November 18, 1998, because he was experiencing such shortness of breath that he could not perform his job duties. (Tr. 120). There is no evidence to the contrary.[11]

Upon consideration of this evidence, which was in existence at the time of the negligence settlements, along with the medical evidence that Claimant had asbestosis and pleural plaques with pulmonary function evidence of both a restrictive and obstructive lung defect, I find that the Claimant was a person entitled to compensation for asbestos related disease. The instant case is similar to the case before the Court in *Cowart*, in that Cowart, already had a loss of wage earning capacity at the time of his 3rd party settlements, although compensation had yet to be awarded.

Therefore, I find that as of the dates of the two settled asbestos cases, the Claimant was a person entitled to compensation within the meaning of § 33(g), and that his claim for asbestos medical monitoring is barred under § 33(g) of the Act.[12]

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[11] Again this finding is made without regard to any subsequent evidence which may have been developed to argue that Claimant's lung impairment, and thus any loss of wage earning capacity, is due solely to COPD and not to asbestosis. The relevant issue is whether, at the time of the settlements, he had a loss of wage earning capacity due to asbestos disease. There is no record evidence at that time indicating that his loss of wage earning capacity was solely due to the COPD, but was instead a combination of asbestosis and COPD.

[12] A finding cannot be made at this time regarding the application of § 33(g) as to any future claim for asbestosis or other asbestos related lung diseases, as the Claimant, for tactical reasons, withdrew his claim for asbestosis upon commencement of the hearing.

COPD Claim

The Claimant has also filed a claim seeking temporary total disability benefits from November 17, 1998 through May 17, 1999 and permanent partial disability benefits from May 18, 1999 to the present and continuing. Claimant asserts that he suffers from occupational lung disease, as a result of his exposure to dust, smoke, fumes and **other airborne particles** during his work as a welder for Employer.

Section 33(g)

Employer also argues that Claimant's claim for COPD is barred under the Act by 33 U.S.C. § 933(g), because Claimant received third-party settlements with asbestos manufacturers without Employer's prior approval. (Employer's brief at 27.)

Section 33(g) specifically refers to Section 33(a) of the Act which states:

> If on account of a disability or death for which compensation is payable under this chapter the person entitled to such compensation determines that some person other than the employer or a person or persons in his employ is liable in damages, he need not elect whether to receive such compensation or to recover damages against such persons.

33 U.S.C. §§ 933(a). Thus, § 33 applies where a third party is liable in damages for the same **disability** for which compensation is sought. In such a case, claimant's right to seek damages from the third party may be assigned to his employer under certain circumstances, *see* 33 U.S.C. §§ 933(b), and where claimant files suit, the employer may gain the rights to a credit for amounts recovered under Section 33(f) and to approve any settlement pursuant to§ 33(g). *See, O'Berry v. Jacksonville Shipyards, Inc. (O'Berry I)*, 21 BRBS 355 (1988), *on recon. O'Berry II*, 22 BRBS 430 (1989); *Goody v. Thomas Valley Steel Corp.*, 28 BRBS 167 (1994).

In this case, there is no dispute that the Claimant's work for this Employer as a welder exposed him on a daily basis to both asbestos fibers as well as other dust, smoke and fumes associated with welding. This is distinguishable from the facts considered by the Board in *Goody, supra*, where there were two distinct injuries (asbestosis and arc welders disease), but where asbestos exposure only occurred while employed by one of the two employers. Here, the Claimant filed two claims for asbestos injury and COPD, both of which are alleged injuries to his lungs. Here also, both injuries are alleged to have occurred as a result of simlutaneous exposure to asbestos and other dust, smoke and fumes associated with welding, all of which occurred while in the employ of this Employer. Thus, it must be determined whether the Claimant's settlements for asbestos disease were for the same **disability** for which compensation for COPD is sought.

The record establishes that the Claimant suffers from both restrictive and obstructive lung impairment as demonstrated by pulmonary function tests, that he has x-ray evidence of pleural plaques, x-ray evidence of asbestosis, and scattered pulmonary fibrosis as indicated by CT scan; and that he has pre-existing asthma since childhood. The Claimant has not worked since

November 18, 1998, when he stopped due to breathing problems. The relevant issue in considering the application of § 33(g) is whether the Claimant's disability due to exposure to asbestos can be distinguished from his disability due to exposure to the other dust, smoke and fumes associated with welding.

In order to establish COPD, Claimant offers the opinion of his treating physician, Dr. Carlos Acosta [13], Dr. George Childs, a pulmonary specialist [14] , Dr. James Baker, another pulmonary specialist who examined and treated Claimant [15], Dr. Eric Freeman, a pulmonary specialist [16], and several pulmonary function tests.

A letter from Dr. Carlos Acosta, dated November 19, 1998, indicates that Claimant was thoroughly evaluated for shortness of breath in 1994 by a cardiologist and a pulmonary specialist. (CX 3-1.) Dr. Acosta states that the final evaluation was that Claimant has severe chronic obstructive pulmonary disease with minimal improvement with bronchodilators. (*Id*.)

A pulmonary function test performed at Sentara Hampton General Hospital on May 13, 1994, indicates a mild obstructive pulmonary impairment which was confirmed by increased RV. (CX 3-18.) It also showed an additional mild restrictive pulmonary impairment. (CX 3-18.) The person performing the exam stated that this opinion was subject to a physician's review. (*Id*.)

The interpretation of a pulmonary function test performed at Sentara Hampton General Hospital on December 29, 1998 indicates a moderate reduction in the FVC and FEV_1 with a normal FEV 1% moderate reduction in the FEF 25-75 and no significant improvement post-bronchodilator; a moderate reduction in the MVV, a mild reduction in the TLC and FRC; a normal RV; normal DLCO; an essentially normal flow loop, except for terminal slowing of the expiratory limb, and a normal ABG. (CX 3-23.) The impressions were, when compared to a previous study performed on 8/5/98, that Claimant's FVC, FEV_1, and TLC was essentially unchanged. Claimant has a combined mild restriction with a small airways dysfunction. The interpreter suggested clinical correlation. (*Id*.)

Dr. Childs' office notes from January 29, 1999 indicate that Claimant was in his office for a follow-up from a previous visit. Dr. Childs indicated that there was no significant change in Claimant's pulmonary function tests. Dr. Childs stated that Claimant had a moderate restrictive defect, but that the Claimant's CLCo was normal. Claimant was sent for a pulmonary exercise study on 1/7/99 because of his complaints. (CX 1-1.) At that time, Claimant was noted to achieve 86% of his predicted heart rate, although he was noted to have a hypertensive response to

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[13] Dr. Acosta is board certified in Family Practice. *American Board of Medical Specialties: Who's Certified* <http:www.abms.org/newsearch.asp>.

[14] Dr. Childs is board certified in Internal Medicine with a subspecialty in Pulmonary diseases. *American Board of Medical Specialties: Who's Certified* <http:www.abms.org/newsearch.asp>.

[15] Dr. Baker is board certified in Internal Medicine with a subspecialty in Pulmonary Disease. (CX 6-40.)

[16] Dr. Freeman is board certified in Internal Medicine with a subspecialty in Pulmonary Diseases. *American Board of Medical Specialties: Who's Certified* <http:www.abms.org/newsearch.asp>.

exercise. (*Id*.) Claimant's maximum oxygen consumption was decreased and he has a normal breathing reserve, but an early anaerobic threshold. Dr. Childs stated that it was the feeling of the interpreter that the findings suggested a possible cardiovascular etiology to Claimant's exercise limitations. (*Id*.) Dr. Childs recommended a reevaluation by cardiologist since there was no evidence of restriction with desaturation which would suggest significant debilitating lung disease.

In his office notes, dated May 7, 1999, Dr. Baker indicated that Claimant's pulmonary function tests demonstrate both a mild obstructive and a restrictive abnormality with normal diffusion capacity. (CX 5-8.) Dr. Baker opined that he did not believe Claimant had asbestosis, but that he clearly had asbestos exposure. (*Id*.) He thought Claimant had an abnormality in his pulmonary function which is compatible with asbestosis, but that his chest x-ray does not indicate pulmonary asbestosis and he does not have any crackles in his chest. (CX 5-8.) Dr. Baker went on to say that he does not know exactly what the etiology of Claimant's exertional dyspnea is, but he thinks it might be the fact that Claimant has a narrow AP chest diameter which might be creating some problems from a cardiac standpoint and the factor that he does have slight restriction in his pulmonary studies. At this point the etiology of that was uncertain in Dr. Baker's mind. (CX 5-9.)

Dr. Freeman states, in his office notes of May 26, 2000, that Claimant was in his office for an evaluation due to shortness of breath. (CX 2-1.) Dr. Freeman stated that Claimant's past lab data indicate that Claimant had a restrictive lung defect, but no diffusion capacity abnormality and no airflow abnormality. (*Id*.) Claimant has a right coronary artery defect, but no pulmonary hypertension, no sign of congestive heart failure or any significant ischemia. Claimant also had an exercise study showing a defect, but the cause was unclear, perhaps cardiac, but that was ruled out by cardiac catheterization. (*Id*.) Dr. Freeman indicates that what is left is the fact that Claimant has a very mild lung restrictive defect of unknown etiology and a lot of symptoms of shortness of breath. He stated that there was very little that could be done unless Claimant's pulmonary function worsened and he developed some type of restrictive defect which can be identified. (*Id*.)

Employer correctly argues that Dr. Baker and Dr. Freeman are both at complete odds with each other over the nature of the Claimant's impairment. Both Dr. Baker and Dr. Freeman have indicated that it is difficult to identify the cause of his lung impairment. Both Dr. Baker and Dr. Freeman read the Claimant's chest x-ray as negative (0/1) for asbestosis. Dr. Baker holds no special qualification for reading x-rays, while Dr. Freeman is a NIOSH B reader. To the contrary, Dr. Harron, who is a Board Certified Radiologist and a B reader, read an earlier x-ray as showing asbestosis (1/1). While Dr. Harron did indicate that the film was of quality level 3 due to overexposure, he did not indicate that the film was unreadable. The opinions of Dr. Freeman and Dr. Baker rely heavily on their opinion that the x-ray evidence is negative for asbestosis. Dr. Baker is also critical of the film read by Dr. Harron, due to its quality and because it is earlier in time than the film he saw, but acknowledges that he did not read Dr. Harron's x-ray. However, both Dr. Baker and Dr. Freeman acknowledge that the Claimant's pulmonary function evidence is consistent with asbestosis.

In weighing the evidence I accord greater weight to the opinion of Dr. Harron that the

film he read is positive for asbestosis due to his superior qualifications as both a Board Certified Radiologist and B reader. Therefore, to the extent that Dr. Freeman and Dr. Baker relied on negative x-ray evidence, I accord those opinions with lesser weight. Upon consideration of all of the evidence, I find that the preponderance of the evidence establishes that the Claimant suffers from asbestosis, asbestos related pleural plaques, and has both restrictive and obstructive lung impairment.

I also find that all of these conditions contribute at least to some degree to the Claimant's impaired lung functioning and combine with his pre-existing asthma. The result is that the Claimant suffers from a single disability caused by his simultaneous exposure to asbestos fibers, smoke, dust and fumes while welding. As such, I find that the Claimant's present disability is the same disability to his lungs which was the subject of the settlement of his asbestos negligence litigation. Because the Claimant settled with the third party asbestos manufacturers, without his Employer's knowledge or consent, in amounts far less than would be payable for even his temporary disability compensation, much less his permanent total disability, I find that § 33(g) bars his claim for COPD.

Because I have found that § 33(g) bars the claim for COPD, it is not necessary to address the issue of § 8(f) relief.

ORDER

Accordingly, it is hereby ordered that:

1. The claims of Willie M. Richardson versus Newport News Shipbuilding and Dry Dock Company for compensation and medical expenses under the Longshore Act are denied; and

2. As compensation is not awarded, attorney's fees are not payable.

A

RICHARD E. HUDDLESTON
Administrative Law Judge